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THE
LARYNGOSCOPE.

VOL. XXXVIII NOVEMBER, 1928. No. 11

ORIGINAL COMMUNICATIONS.

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THE DIAGNOSIS AND LOCALIZATION OF INTRACRANIAL LESIONS. CONSIDERED AS A RESEARCH FIELD. PRESENTATION OF CASES.*

DR. MARVIN F. JONES, New York.

The rapidly increasing interest in the diagnosis and localization of intracranial lesions is the natural result of curiosity and ambition.

We have seen stimulation of the inner ear produce reactions in numerous parts of the body. By irrigating the ear with cold water we note nystagmus, loss of balance, incoordination of muscle groups, pallor, gastric disturbance and mental reactions. It would, indeed, be strange if these reactions did not invoke our curiosity to the point of investigation. They bring us into a new and rather unknown field, but it is possibly all the more attractive because of this.

Various investigators have studied the physiological reaction in response to stimulation of the labyrinth, others have studied the pathways over which these reactions travel to produce general phenomena, and still others have tried to interpret from a distortion of these phenomena where the lesion is located that caused the abnormal response to the stimuli. These investigators are enabling the physician, practicing the head specialties, to aid materially in the diagnosis of intracranial conditions. It is, at present, insufficient to limit our reports to the ear pathology and now we should add our interpretation of localizing symptoms.

*Presented as Candidate's Thesis to American Laryngolocal, Rhinological and Otological Society, Washington, D. C., May 3, 1928.

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Nearly all physicians practicing ophthalmology, rhinology or otology have had a basic training in all these divisions. It is a natural conclusion that the specialist should have a much more intimate knowledge of the anatomy, physiology, histology and pathology of the head, with the possible exception of the cranial contents, than any other physician.

One of the greatest aids in precise localization of intracranial lesions is through involvement of the 12 cranial nerves or their centers. The first nerve, or olfactory, is infrequently mentioned as an aid to diagnosis, but references are made to disturbances in the sense of smell when the intracranial pressure has been sufficeint to cause these disturbances, or when the lesion has eroded the tract itself. There is a fertile field for research in this direction.

The second, or ophthalmic, nerve is very frequently involved and this is manifested by choked disc, contraction of form fields, irregularity in the color fields and complete or partial loss of visual fields.

The third, or oculomotor, nerve is peculiar, in that individual muscles or the entire group supplied by it may be affected by central lesions. Paralysis of the levator palpebrae is possibly the most common individual pathological condition observed. The superior rectus, inferior rectus and inferior oblique are included in a case presented later in this paper.

The fourth, or trochlear, nerve is seldom mentioned as being involved and it is difficult to determine whether this is due to lack of accurate observation or the difficulty in diagnosing a paralysis of the superior oblique muscle.

The fifth, or trifacial, nerve is a combined sensory and motor nerve which is joined later by sympathetic branches and has a complex ramification. The symptomatology produced by a lesion involving this nerve is sometimes most enlightening. For example, one of the early symptoms of an eighth nerve tumor is anesthesia of the cornea. Pain, paresthesia or anesthesia over the distribution of its three main branches is quickly noticed by the patient. Irregularity in the action and size of the pupils also gives evidence of involvement of this nerve.

The abducens, or sixth, nerve seems to be particularly susceptible to any intracranial lesion, especially a lesion involving the base. Its long pathway through the cranium, from the junction of the lower border of the pons and the upper portion of the medulla, to the superior orbital fissure may account for the frequent involvement. External rectus paralysis with diplopia is a most common early symptom of intracranial lesion.

The seventh or facial nerve furnishes the most striking symptoms observed in these lesions. The assymmetrical, expressionless, paralyzed face is most distressing to the patient. While in itself it is not serious, it is too often an indication of a central pathology.

The eighth, or acoustic, nerve, with its double function of audition and equilibrium, furnishes the most information of central disorders to the otologist. The importance of the eighth nerve will be emphasized in the reports of cases.

The ninth, or glossopharyngeal, a combined motor and sensory nerve, carries sensation from the mucous membrane of the mouth and the special sense of taste from the posterior portion of the tongue. Also, it supplies motor filaments to the pharynx, tongue and mucous glands of the mouth. It is closely associated with the tenth and eleventh nerves, so that many of its responses are due to a combined action of the three.

The tenth, or vagus, is the only nerve that has a major portion of its function away from the head. Even the tenth, however, has a distinct laryngeal function in the movement of the constrictor pharyngis inferior and all the muscles of the larynx except the cricothyroideus. It also supplies sensory fibres to this area. It contributes an auricular branch and is the principal motor nerve of the pharynx.

The spinal accessory, or eleventh, nerve acquires most of its laryngeal function through its anastomosis with the vagus.

The twelfth nerve, or hypoglossal, has the motion of the tongue as its principal function of interest to laryngologists.

It will be observed from the above enumeration of the cranial nerves that the function of each concerns particularly our specialty. The disturbance of these functions furnishes the most accurate means of localizing a large percentage of intracranial lesions.

All medical practitioners realize that the percentage of error in the diagnosis of intracranial lesions is high. It is also recognized that most of the correctly diagnosed cases are advanced cases. The hope for the future would then be in the direction of more accurate and earlier diagnoses. Consider what otology alone has offered to attain this goal.

The following references are taken from the Transactions of the American Otological Society since 1897.

1. WILSON and PIKE: Experimental Work on Animals, Producing Lesions of the Cerebral Hemisphere. Vol. 13, Pt. 2, 19??

2. WILSON, J. GORDON: Differential Diagnosis of Lesions of the Labyrinth and Cerebellum. Vol. 13, Pt. 3, 19??

Among other statements, these important differential points were claimed:

A. Variations in the attitude of the head influence, the lack of equilibrium of the body in labyrinthitis and not in cerebellar lesions.

B. An affection of the labyrinth does not definitely involve those movements of isolated parts which result in dysmetria.

3. RANDALL and JONES, ISAAC: The Ear Tests of Baranay in Locating Cerebellar and Other Encephalic Lesions. Vol. 13, Pt. 3, 19??

In this they state:

"We can usually differentiate lesions of the labyrinth from those of the cerebellum." "We can always tell when the eighth nerve is diseased. We can state positively when the posterior longitudinal bundle is affected. We can detect a lesion of the cerebellum, but cannot always locate it."

4. WILSON, J. GORDON: The Relation of the Ear to the Central Nervous System. Vol. 14, Pt. 1, 19??

In this paper he urged a closer cooperation between the neurologist and the head specialist to facilitate the study of a subject common to both.

5. EAGLETON, WELLS P.: Vol. 15, Pt. 3, 19??

As a presidential address that year, Eagleton presented a series of cases dealing with intracranial surgery on cases with a primary ear involvement.

6. INGHAM and JONES, I. A.: Vol. 15, Pt. 3, 19??

Discussed *neurologic problems* and in their conclusions made these statements:

A. Spontaneous vertical nystagmus in patients who have other symptoms of intracranial lesion indicates that the lesion is located within or adjacent to the brain stem.

B. Perverted nystagmus following ear stimulation, is an indication of involvement of the brain stem.

C. Tumors of the cerebellar pontine angle frequently show loss of auditory and vestibular function on the same side as the tumor. There is also absence of response from the vertical canal of the opposite side.

The May, 1925, meeting produced an evidence of marked increase in the interest in neuro-otology. There was also evidenced a decided diminution in the mortality rate following operations on intracranial complications of otologic pathology.

7. WISHART, D. E. S.: Report of Neuro-otologic Findings in Seven Consecutive, Verified Cases of Brain Tumor in the Posterior Fossa in Children. Vol. 17, Pt. 1, 19??

8. CAHILL, HENRY P.: Presented Nine Cases of Cerebral Abscess and Three Cases of Cerebellar Abscess, with 100 Per Cent Recovery. Vol. 17, Pt. 1, 19??

He attributes these results, in a large measure, to the use of the Mosher drain. At this same meeting, Lewis Fisher has this paragraph as the ending of his paper:

"In most of the cases the neurologic studies disclosed the true nature of the case. It is interesting, however, that the neuro-otologic examination, made independently and without knowledge of the neurologic findings and conducted

by otologists unskilled in neurology, reached the same conclusions as shown by the above figures. What is even of greater importance is the fact that in a number of cases, of which the reported ones are an illustration, the otologic examination was the only method of study by which the diagnosis could be made."

When all neurological symptoms are present it is not difficult to diagnose and localize intracranial lesions, but in the early stages these characteristic symptoms are lacking. When a tumor causes gross impairment of function, it has reached the advanced stage. If stimulation of the vestibular apparatus produces reactions in the sensory, motor and sympathetic systems, an early interference with these reactions may be elicited. The recent research in this direction has awakened the otologist to the possibility of employing the vestibular reactions in early intracranial diagnoses. Among the more prominent workers in this research have been Magnus and De Kleijn, Tait and MacNally and Wilson and Pike.

Probably every otologist has had under his observation the so-called "atypical reaction' 'to ear stimulation. For example, a patient will respond normally to a caloric test excepting the nystagmus. This may be absent or perverted. Some have perverted falling reactions, with all other findings normal. Others have an atypical pastpointing with normal nystagmus, falling, sensation of falling and sympathetic reactions. As long as we find presumably normal individuals reacting in a regular manner, we may presume that those patients who do not react in this manner are abnormal. The most plausible location for this abnormality is somewhere in the reflex arc between the ear and the reacting organism.

The following summarized cases have been chosen at random from many similar ones. They illustrate three abnormal pastpointing reactions.

Miss E. L.: The following is a case of normal pastpointing with the left hand and no pastpointing with the right, following caloric, and crosspointing following rotation. The history is that she has dizziness, sensation of falling forward and objects seem to rise. These attacks have made it necessary for the patient to go to bed for days. They are brought on by quick motion. Physical examination is negative. Has running ears occasionally.

Rotation Tests: Rotation to the right (normal nystagmus), right hand pastpoints to the right and left hand to the left. Rotation to the left-right hand pastpoints to the left and left hand to the right.

Caloric Test: Nystagmus normal after irrigating the right ear—no pastpointing with the right hand—left hand pastpoints to the

right. After irrigating the left ear—no pastpointing with the right hand—left hand past points to the left.

Miss E. H.: The following case is an example of absence of pastpointing following caloric test of one ear and normal pastpointing following stimulation of the opposite ear. Vestibular apparatus functioning on both sides. Nystagmus normal both sides. History is that patient has had rumbling noises in his ears at times, which seem worse during damp weather, never had an earache but has had discharging ears—rumbling attacks following every meal. Has had gall bladder operation but vomiting still persists. Headache mostly on right side, which comes on any time during the day, when severe lasts for days but usually only 24 hours. Has dizzy attacks but does not fall.

Examination of Ears: Left ear: there is old healed perforation superior to short process and anterior to Shrapnell's—dry at the present time.

Right ear: almost identical findings—perforation in this ear seems to have slight amount of secretion. All other findings are negative—slight spontaneous pastpointing to the left with right hand—tendency to pastpoint to the right with he left hand. Roation reactions are no sufficiently marked to be outside of normal.

Caloric Test: Normal nystagmus from stimulation of both ears—when the right ear is stimulated there is pastpointing to the right with both hands—no pastpointing after stimulation of the left ear.

Mr. F. S.: The following is a case which is an example of not infrequent occurrence; with all other findings within normal limits, he had no pastpointing following caloric test.

History: About five years ago first noticed sizzling noise in his left ear—this was quiet for a while, but for the last month-and-a-half has been more marked. He becomes dizzy when walking, has a feeling of pressure in both ears and sensation of rushing air in the left ear when lying down. During the dizzy attacks has swaying sensation but does not fall, especially marked when patient looks up or down. Objects in the room are stationary. The feeling is lack of balance, feels as though the ground was coming up to meet him, also has sensation of pressure in back of head.

All examinations are essentially negative. After rotation pastpointing is normal—nystagmus is normal, but after caloric test has normal nystagmus but absence of pastpointing.

It is impossible to tell what the interference is or where it is located, yet these are abnormal reactions in patients giving evidence

of an organic pathological condition. The only way to prove this assertion is through postmortem examination.

A field exists which demands the combined experience of neurology, otology, ophthalmology and rhino-laryngology. It is a fertile field for research. It should not be considered as oto-neurology but as a combination of the head specialties and neurology. Of the head specialist it demands a thorough preparation in a small portion of neurology, combined with an intimate knowledge of brain anatomy. The cases presented are those having a history combining symptoms referable to neurology, ophthalmology and oto-laryngology.

Examined Jan. 25, 1927.

Family History: Negative.

Past History: He had two to three months discharge from his right ear and dizziness during the war. Radical mastoid operation eight years ago. This was followed by profuse discharge for three to four years—it resolved and three or four times in the last year it started discharging again. About four years ago had fever and both sides of his face swelled. Tonsils removed 12 years ago. Last July had a secondary removal and it took three days for him to recover. Had a real convulsion at this time. He is usually not the least bit nervous but has become irritable.

Present Illness: Has had subnormal temperature of about 1½° for the past week and has been dizzy. Some nausea and vomiting, uncertain feeling and light headedness, which make his gait irregular. Appears lethargic—rather scanning speech. Usually vivacious, but recently cannot keep awake. He has become more irritable recently according to his wife's statement and this following minor occurrences. Ordinary disposition has been exceptionally good. Headache on the right side—yesterday afternoon he had a dull headache over the right side, which lasted until 11 p. m. Seldom falls in any particular direction, but after this headache he felt as if he were falling over his plate following dinner. He had a severe shaking attack at the office which resembled a chill. Objects in the room have no regular rotation but oscillate. Previous to operation had had this sensation of objects whirling around—could not get out of bed and walk at this time. He is unable to sleep well at night, but is quiet and without convulsions. Pulse has been between 60 and 70. When he has vomiting attacks, he will gag two or three times and then vomit—not projectile type.

A. Examination: Eyes: very fine spontaneous nystagmus to the right, which is transient, present to the left, but not as marked. (There is no spontaneous pastpointing in either hand. Diminished

right reflex; left active. His abdominals are more active on the right than on the left. Left are present.) Pupils react to L and A; fundi negative.

B. Right ear shows result of former radical mastoid operation, which evidently was well done, and there is no discharge at this examination. Left ear: slight tubal congestion and retraction of membrana tympani.

C. Nose negative.

D. Throat negative.

E. No spontaneous pastpointing; negative Romberg. Irrigation, right ear, water 68° C., head erect, horizontal nystagmus to left appeared after eight minutes' duration, with the head posterior, horizontal nystagmus to left with very wide excursions. Quick and slow components both in normal ratio. Pastpointing to the right with both arms; tendency to fall toward the right. Romberg not tried, due to instability of patient to stand; would undoubtedly have been to the right.

SEGMENTAL REFLEXES	R.	L.	SUPRA SEGMENTAL REFLEXES	R.	L.
Pectorals	Active	Active	Abdominal:	More active than left	Diminished
Bicepitals:	Dim	Active	Plantor:	Normal	Hyperactive
Tricepitals:	Active	Active	Kernig:	Not present	——
				No deviation of tongue	
Patellars:	Present but dim	Active		No involvement of 7th	
Achilles:	Dim	Active			
Clonus:	No ankle clonus either side. Patellars more marked when lying down. Left exag.?				

Laboratory Findings: Jan. 31, 1927, X-ray examination shows no abnormality in illumination detail or outline of the accessory nasal sinuses. In the central region an extensive area of rarefaction extends along the pericementum of the upper left lateral incisor. Further examination will be necessary when patient is in condition.—Dr. Wm. H. Meyer.

Jan. 37, 1927, X-ray examination shows no gross abnormality in position, detail or outline of the lumbosacral spine.—Dr. Wm. H. Meyer.

Report on Examination of Blood: Jan. 25, 1927: Age 31 years, sex male. Leucocytes 7,500 per c.m.m. Differential count of 100 white cells; polynuclears 73, lymphocytes 27.

Report of Examination of Spinal Fluid, Jan. 39, 1927: *Physical Examination:* Color, clear; translucence, clear; volume, 5 c.c.; specific gravity, Q. N. S. *Chemical Examination:* Butyric acid test, negative. *Microscopic Examination:* White cells per c.m.m., none.

Cultural Examination: Diagnosis not made, due to lack of cells and globulin. Wassermann negative.

Jan. 28: Examination of the right ear revealed a small amount of thick, purulent discharge with foul odor, apparently coming from the epitympanic space. As the patient was rapidly becoming more drowsy an exploratory operation for a possible brain abscess was considered advisable.

Neurological Examination, Jan. 28: Patient alert, but seems slow in respon es. Cranial nerves: Pupils, L and R, 4 m., 7, 3.5 m.m. Both react to light promptly, irregularity in outline. Palpebral fissures, L greater than R. Diplopia occasionally when light is held slightly to left of midline. Weber test referred to right. Reflexes, biceps both diminished. Triceps, both active and equal. Patellars, L greater than R slightly. Achilles, L greater than R slightly. Abdominals, L greater than R. Sensory, normal except small patch on lower temporal region on right. Fundi are normal. Swelling on left maxillary region, tenderness on lower lumbar region. *Diagnosis:* 1. Possible pachymeningitis right. 2. Possible brain abscess right temporosphenoidal region.—Dr. Blakesley.

Diagnosis: Possible brain abscess.

Operation: Mastoid cavity and middle ear cleared of granulations. Necrotic area in tegmen tympani and small amount of cholesteatoma removed. Surrounding area of dura exposed. No adhesions or visible pathological changes in dura. Exploration of brain substance not considered advisable. Wound closed.

Progress Notes: Feb. 1, 1927: Dressing clips removed, wound good condition, no pus, patient apparently doing nicely. Feb. 4: The fundus findings are the same as found prior to last operation; namely, myopic conus in left fundus. There is no elevation of the discs and no vessel changes.—Dr. Seager. Feb. 11: Neurolagical examination: pupils about 5 m.m. in diameter, react to light promptly, palpebral fissures seem equal, no diplopia today. The reflexes show left patellar slightly more active than right. Left abdominal slightly less active than right, all other reflexes are active and equal. Bilateral reflexes. Fundi are normal, rather hyperemic.—Dr. B. Wheeler.

Feb. 12, discharged, condition appears good.—Dr. Campbell.

Oct. 14, patient at work, apparently well, ear dry.

Comment: The symptoms in this case would indicate a brain abscess. At the time of operation it was decided to do a secondary operation and explore the brain through a temporal trephine. As

the patient improved rapidly and his symptoms later entirely disappeared, no further operative procedure was deemed advisable. There is a possibility that the abscess is still present.

B. Y., age 5 years, admitted June 16, 1927, Babies' Ward, New York Post-Graduate Hospital.

Family History: Rheumatism, mother for one month two years ago. Grandmother had tuberculosis and died before birth of patient.

Past History: Development normal, measles at 4, occasional sore throats, glands, snores, mouth breather.

Present Illness: Chief complaints, pain in ear, discharge for three days. Headache for one week.

Three months ago had diphtheria, given antitoxin, was well for three weeks, then developed scarlet fever, mild attack, temperature for three days, 102° highest. There is a trace of albumin in urine during illness, but no other complications. Developed chickenpox during quarantine six weeks ago and was well until one week ago, when she complained of pain in left ear; had fever that night and next day discharge appeared in the ear. Headache began then and pain was severe and constant; this was present in the forehead. Strabismus of both eyes noted at the same time and diplopia complained of. Vomited all food or fluid for four days, occasionally vomited without relation to intake food; this has stopped at present time. Headache is still present, temperature ranges from 100° to normal, had normal temperature four days ago, which has continued up until the present time. Pain in the ear still present. For four days had a slight twitching to the right side of face, occasionally for one day. Appetite is very poor; bowels move by enemas or cathartics. Has been very restless, agitated, talkative when no pain was present. Slept poorly the first three days but is now sleeping better. Lumbar puncture was done two days ago, increased pressure, normal fluid, no other results given. Impression of first examination by admitting doctor was: *1.* Encephalitis lethargica. *2.* L. O. M. C. A. *3.* Mastoiditis. *4.* Brain abscess.

Physical Examination: A. Eye examination: apparent weakness of the external recti, no real strabismus but rather alternating esophoria. Visual fields both slightly contracted; not marked. *B.* Ears: right drum negative. Left ear discharged, drum thickened and opaque, landmarks absent, not visible, large ventral rupture, posterior wall bulging forward slightly (examination by house surgeon). *C.* Nose negative. *D.* Mouth negative. *E.* Tonsils very large and cryptic, tongue slightly coated. *F.* Neck, chest, heart, lungs, abdo-

men, genitals negative. *G.* Weakness of extremities but not paresis or paralysis. *H.* Lymph glands, posterior cervicals enlarged.

Progress Notes: June 16, 1927, child has had a history during the past four months of one acute illness after another. This, I think, with O. M. P. A., might account for her present asthenia and apathy. There are no positive abnormal neurological findings at this time. Advise ear consultation.—Notation by Dr. Sherwood, Neuroolgist.

June 17, there is an extreme bilateral papillitis with swelling and tortuous veins, very dark appearance of blood in veins, retina appears very hyperactive. Weber referred to the left ear, headache is frontal and over left mastoid region. Left palpebral fissure diminished and there is a left facial weakness. Advise X-rays all sinuses and left mastoid. Consultation to Eye Department. — Drs. Blakesley and Sherwood.

June 18, fundus examination: swelling of both discs at 5 diopeters elevation. Diagnosis: Possible brain tumor.—Prof. Cohen and Enenfeld.

June 18, canal wall sagging; left ear, possible mastoid, tenderness over tip and some bulging at Shrapnell's membrane.—Notation by House Surgeon, Dr. Seagar.

June 18, lumbar puncture, fluid under moderately increased pressure, clear, cell No. 3, three lymphocytes, no increased globulin.

June 20, there was delay in the consultation slip reaching the Otological Department, consequently the child was not seen until June 21; the request was made on June 16. At this time the right ear appeared normal; left ear, bulging membrana tympani, very small amount of discharge, no prolapse of the canal wall, impression was given of resolving drum. No marked tenderness over the tip, advised myringotomy as a precautionary procedure; myringotomy performed, pus discharge followed. There was no spontaneous nystagmus, no paralysis of eye muscles, facial weakness was not noted and tongue appeared normal.

June 21, small amount of purulent discharge from the left ear, otherwise condition unchanged.

June 22, refraction of retinal vessels is normal, but a plus four is neecssary to see the nerve head. Margins of the left disc are beginning to appear more distinct than at previous examinations.—Prof. Cohen.

June 23, right eye: numerous fresh flame-like hemorrhages bordering on the upper and lower margins of disc, elevation same as previously. Left eye: numerous large hemorrhages bordering uppper

and lower margins of disc. Swelling same as previously. These findings will incidate an increase in the pathological lesion.—Notation by Dr. Cohen.

June 23, discharge from the ear evidently diminished and the child appears bright and happy.

June 24, discharge still diminishing, but on account of the increase in the papillodema, it was thought advisable to do an exploration on the mastoid, and this was done the same afternoon.

At the operation usual incision was made and the periosteum retracted; cortex appeared dark and hyperemic. The cortex was opened with a curette with an immediate outflow of pus under some pressure. The entire mastoid cavity was eroded and filled with granulations. The sinus wall was almost completely eroded near the bulbar end and the necrotic process extended posterior into the bone over the cerebellum. Rather considerable cerebellar dura was exposed and dura covering cerebellum and sinus was tremendously thickened. There was no line or evidence of a demarcation between the sinus and the cerebellar dura. Probable location of the sinus was opened by an incision posteriorly along the sinus wall until a clot was found over the torcular end, which was removed, and free bleeding occurred. No free bleeding occurred from the bulb end.

Child at this time was in poor condition so it was decided to do a ligation of the jugular vein instead of a resection. This was accomplished but was rather difficult owing to the collapsed condition of the vein. Neck wound was closed and the mastoid wound partially closed. The child's condition at this time was exceedingly poor, radial pulse not palpable and the child was cyanosed. Anesthesia had been stopped some time previous to the neck incision. It was thought advisable to give an infusion of saline and this was attempted, without success. Repeated hypodermics of Spiritus frumenti were administered, together with one dose of digitalis. The pulse became palpable, the child in slightly better condition. At various times the pulse would fall and the child received injections of whiskey. Immediate transfusion was considered essential and this was done the same evening, 500 c.c. direct transfusion by Dr. Unger, using the Unger method. The child was returned to the ward and seemed to react well from the transfusion.

Laboratory Examinations, June 20, 1927: X-ray examination of the accessory nasal sinuses shows insufficient evidence of pathological change to be of Roentgen diagnostic value. It is worthy of note, that as usual at these years, no pneumatic frontal locules exist; there

being only supraorbital extensions of the ethmoids on the left side. A further examination shows a clouding over the left mastoid, suggesting an exudative infiltration. The mastoid area is small, with, however, small pneumatic locules visualized. A detail of the septal divisions appears preserved, suggesting as yet no evidence of necrosis.—B. W. J.

Negative for sinuses, exudative mastoid.—Dr. Wm. H. Meyer.

June 23, 1927, X-ray examination of the skull shows no gross abnormality in detail or outline of the bones of the vault or face, excepting perhaps a slight accentuation of the paccionian depressions, more noticeable in the middle fossa.—Dr. Wm. H. Meyer.

Slight accentuation paccionian depressions, middle fossa.—B. W. J.

Urine was persistently negative. Blood count, June 17, 1927, leucocytes, 24,400; June 18, leucocytes, 11,800; polynuclears, 69; lymphocytes, 22. Spinal fluid was under increased pressure before admission, otherwise negative. Culture from the mastoid showed pneumococci.

Progress Note: Oct. 14, 1927, patient in apparently good health, mastoid and neck wounds healed, ear dry.

Comment: The child's condition was apparently improving just preceding the operation. The temperature was normal; discharge in ear diminishing following recent myringotomy; tenderness over the mastoid had disappeared and the child was playing in bed. In view of the increasing intraocular pathology, however, an exploration of the mastoid process was thought advisable.

C. M., age 28 years, male, black, painter; admitted to New York Post-Graduate Hospital May 8, 1927.

Family History: Wife had a spontaneous miscarriage at four months, later gave birth to two healthy children.

Past History: Tonsillectomy, 1924; gonorrhea, 1921.

Present Illness: May 1, 1927, headache starting at night, located over the right frontal region, and on May 3 became general. Sore throat and painful swelling. On May 2, 1927, pain in right ear, pain in the right eye, diplopia and dizziness. Chill on May 3, nausea. May 4, vomited three or four times, vomiting of food recently eaten. May 4, staggering gait, with tendency to fall and walk to the right. Appetite poor, sleeps poorly, feels dull and irritable. He had not been confined to bed.

There was spontaneous pastpointing to the left with the right hand, no pastpointing with the left hand. Followngi irrigation of the left ear, there was a fixed stare to his eyes, blanching of the lips. When

asked to look at the examiner's finger and the position of the finger was changed from one point to another, the eyes would not immediately follow, but later would jerk into focusing position. Nystagmus was same in the vertical as in the horizontal position. Pastpointing not changed. Left ear: no nystagmus after 1 minute 20 seconds' irrigation; patient showed symptoms of sympathetic involvement and the irrigation was stopped. There was pastpointing to the left with both hands following this irrigation.

Eye Examination: Discs showed no elevation and no haziness. Slight paresis of the left external rectus. Spontaneous nystagmus, horizontal to left and vertical. Diplopia present.

Neurological Examination, May 9: Appendicular co-ordination tests are normal, pupils irregular and unequal, probably due to eye medication. Light and convergence reflexes sluggish, superficial and deep reflexes unequal.

Diagnosis was acute infection involving the eighth nerve and also the sixth nerve of the left side, primary focus being in the throat. Epidemic encephalitis must also be considered. Neurological examination was made by Dr. Blakesley.

Laboratory Findings: All tests on spinal fluid negative for syphilis, Wassermann negative. Blood count, May 10: leucocytes, 5,100; polynuclears, 80; lymphocytes, 20, mononcular leucocytes, 4; small lymphocytes, 16. X-ray of all sinuses negative.

Patient with the head forward falls to the right, with the chin over the left shoulder falls forward, chin over the right shoulder falls backward.

Progress Notes: Patient under ordinary physical care, plus local treatment to the throat, made a slow, complete and uneventful recovery.

Vomiting, spontaneous nystagmus, vertical nystagmus, paresis of the external rectus, diplopia and the hyperactive patellar on the right and the sluggish abdominal on the right, spontaneous pastpointing, falling to the right and walking to the right would lead us to suspect a central lesion. The results of the caloric tests, combined with the falling on changing position of the head lead us to believe there was a toxic condition either of the labyrinth or the nerve, and these toxemias of the acute types sometimes do affect the ocular muscles.

R. A., age 9 years, admitted June 29, 1925. Date of birth unknown.

Family History is negative. *Past History* not essential.

Chief Complaint: Some pain in and back of the left ear, profuse discharge of pus from the left ear.

Present Illness: Started about two months ago, when she had a discharge in the left ear without any marked pain or constitutional symptoms. This continued without other symptoms until Saturday night, June 27, when she began to cry with pain in her left ear. Pain has been almost continuous since. Sunday she was seen by a physician and he reported that she seemed somewhat delirious. She was again seen Sunday night, when temperature was 105°. She vomited that night and again Monday morning, June 29, when she was admitted to the New York Post-Graduate Hospital.

Examination of the Ears disclosed tenderness over the left mastoid region, profuse discharge, prolapsed canal. She was an extremely sick-appearing child. An immediate mastoid operation was advised. She was taken to the operating room the same day and the usual mastoid operation was done, uncovering the sigmoid sinus. On palpation, the sinus did not fluctuate and a diagnosis of thrombosis was made. The sinus was exposed throughout its entire length and an incision in the sinus wall was made. Bleeding was free from the torcular end, but only a slight amount of bleeding from the bulbar end. There was a thrombus on the wall toward the bulbar end. Ligation of the jugular vein was done and the child was given 500 c.c. normal saline infusion while on the table. Patient was typed for a transfusion. This was given as soon as possible. Another transfusion given July 2 and a third given July 6.

Neurological Examination was negative following operation. Child too frightened to co-operate for the complete examination. No pathological signs of central nervous system involvement or meningitis. Lumbar puncture was suggested if patient did not improve. July 6, patient lies on the left side, distinct decrease in the tonus of the left upper and lower extremities. Left hand droops, diadokokineses of the left hand. Reflexes, deep and superficial, active and equal. These examinations were done by Dr. Blakesley. July 7, spinal puncture; about 10 c.c. clear fluid under moderate increase in pressure. Some increase in globulin. July 8, ophthalmoscopic examination shows both nerve heads to be normal in color, sharply defined margins. Retinal vessels slightly engorged. This would not indicate intracranial pressure.—Dr. John H. Dunnington.

Because of the neurological findings and the general condition of the patient, it was decided to uncover the cerebellar dura and possibly to explore the cerebellum. The child was prepared for operation and the cerebellum below Trautman's triangle and posterior to the sinus was exposed. Cerebellum was under tension and bulging. On the advice of Dr. Maddren, a brain trocar was inserted into the cere-

bellum to discover supposed abscess. No pus was found; there was considerable exudation of cerebrospinal fluid. Child was sent back to the ward in very poor condition and grew rapidly worse. On July 12 there was a hypodermoclysis of saline and glucose given. Patient comatose and died the same day without regaining consciousness.

Postmortem Notes: All attempts made to obtain a necropsy unsuccessful.

Laboratory Findings: Cell count was 43 white cells per c.m.m., increased globulin. Cultural examination was negative. July 6, 1925, blood culture showed one colony on one plate which may have been contaminated, diagnosed streptococcis viridans. Urine examination July 6, large amount of diacetic acid. Blood culture, July 7, was negative. July 7, report on mastoid showed streptococcis viridans and Gram negative bacillus present. Spinal fluid examination, July 7, negative for syphilitis.

S. P., age about 38 years, entered New York Post-Graduate Hospital May 26, 1927, with complaint of paralysis of the left side of his face and weakness of both legs.

Family History was negative. *Past History:* Rheumatic attack, with shooting pains in both legs, occasional sensation of something crawling over both legs. He had discharging ear during the war for three months; this resolved and aside from a slight difficulty in hearing, he had no further complaint until the present illness.

Present Illness: One month ago the patient began to have headaches. This was followed by drooping of the left eyelid, which became complete in three days. One week later he developed left facial paralysis; this became complete about two weeks before admission.

Examination: There is a complete left facial paralysis, without pain, and apparently he is not acutely ill.

Eye Examination: Ptosis of the left eyelid, left pupil does not react to light and is slightly irregular. Right pupil reacts slightly to light, corneal anesthesia right, second degree spontaneous nystagmus to left, which is noticeable in the right eye. Paralysis of the superior rectus, the inferior rectus, the internal rectus, superior oblique and inferior oblique muscles of the left eye.

Otological Examination: Deafness in the left ear, small perforation of the membrana tympani, which is superior and anterior. A small probe was passed through this for a short distance along a sinus, upward and forward. The ear was dry at this examination.

Left ear: irrigation with water at 68° after 40 seconds produced a rotary nystagmus to the right, which was about 20 seconds' duration. There was no reaction from the horizontal canal. He had a dizzy sensation, but no pastpointing, following irrigation. The functional hearing tests on this case at the first examination were lost, but from memory the high tone limit was reduced, the low tone limit elevated, bone conduction reduced and the Weber referred to the right ear. The right ear was not tested on account of the objection of the patient.

Nasal Examination was negative.

Mouth: Tongue was slightly coated and deviated to the right. There was a weakness of the soft palate of the right side.

Neurological Examination shows a positive Romberg, with a falling to the right, ataxia on the right side, heel to knee test very ataxic on the right, to to object above the patient, right ataxic, left normal. Heel to knee along shin atavic on the right, left normal, adiadokokinesis on the right. Biceps, triceps, radials, superpatellar and patellar more active on the right than on the left. Abdominal reflexes less active on the right. There is a questionable Babinski on both sides, pain and temperature sense normal, pain and muscles sense normal.—Neurological examination by Dr. Blakesley.

Consultation slip was sent to the Otological Department immediately after a spinal puncture was taken and a note that the findings would be unreliable following this procedure was made on the chart. This consultation slip was sent to determine if the eighth nerve was involved and if the seventh was secondary to the ear condition or part of the general findings. From the functional examination and caloric tests it was decided that the eighth nerve had been involved, and since the involved ear had been resolved for 10 years and paralysis of the seventh nerve was rapid in its development, there was no connection between the ear findings and the involvement of the facial nerve.

Laboratory Findings: Complement fixation test on the spinal fluid for syphilis was positive, all other laboratory findings were negative.

Diagnosis: Luetic basilar meningitis which involved the third, fourth, upper division of the fifth, seventh, eighth, and possibly the twelfth nerves, was made. Under antiluetic treatment the patient has improved. This case was tentatively diagnosed. A basilar lesion, probably gumma or tumor, and the upper portion of the fourth ventricle, with a separate involvement of the seventh nerve, due to the ear. With positive involvement of the eighth nerve and no

observable cause of the seventh, the diagnosis was finally agreed upon as luetic involvement of the meninges and nerves of the base. Complete resolution by giving antiluetic treatment.

Lumbar punctures, except in cases of emergency, should be done after all other examinations have been completed. Oto-neurological tests will be completely changed and rendered without value following this procedure.

M. L., age 4 years, 4 months, was admitted to the New York Post-Graduate Hospital March 10, 1927; born in Russia.

Family History: Two cousins with spontaneous congenital nystagmus. *Past History:* Normal birth and development, had measles at 2 years of age, recurrent sore throat and aphonia.

Principal Complaint: Discharge from the ears with fever, about Jan. 15 left ear began discharging pus; soon after this the right ear pained and was incised. Both ears have discharged since that time and the patient has been running a constant temperature. Degree of temperature was unknown to the mother.

At the time of admission to the hospital diagnosis was double mastoiditis with marked impairment of hearing. At that time the examination showed horizontal, slow nystagmus to the right and left, rhythmical, with long, undulatory oscillations. This was constant. Tongue was coated, tonsils enlarged and inflamed.

Examination of the abdomen revealed an enlarged spleen and slightly enlarged liver. Reflexes were exaggerated, with the right more pronounced than the left. There was a questionable Babinski, cervical and inguinal glands enlarged. There is an eczematous eruption around the buttocks.

Neurological Examination, May 10, 1927: Child has a horizontal nystagmus, with a rapid component to the right, muscles of the right upper and lower extremities show increased tone and the reflexes on the right are more active than on the left. There is a positive Babinski on the right. The abdominals are less active on the right than on the left. Romberg is negative. There is no ataxia on walking. These findings and symptoms are best explained, I think, by an epidural irritation on the left side due, no doubt, to advanced mastoid destruction. This neurological examination was made by Dr. Sherwood.

Ophthalmic Examination: Disc margin well defined, no evidence of papillitis or papillodema. Mastoidectomy, bilateral, performed March 10.

Progress Notes: May 14, 1927, condition of the mastoid wound was satisfactory, two clips removed on either side, nystagmus present and fully as active as previously. May 25, mastoid wound in excellent condition.

Examination of the Eyes, May 29: The reason for this examination was that there had been a slight irregularity in the temperative curve and the child apparently was not as well as it had been. Eye examination by Dr. Alger showed media clear, fundi normal, and he makes a note that the nystagmus prevents satisfactory fundi examination. Nystagmus resembles the congenital central type. I was called for an otological examination at the same time Dr. Alger examined the patient and found a tendency to fall to the right on walking, marked atonia of the right arm, and diadokokinesis of the right side. Head tipped to the right in a postural position. Reflexes: a distinct Babinski of the left side, an increased patellar reflex. The child was observed for a few days, and as the temperature became normal and symptoms diminished, he was treated expectantly. The child was discharged from the hospital, April 5, 1927, with the above findings, normal.

Laboratory Findings: Urine, upon repeated examinations, remain persistently negative. Blood count, May 11, 1927, leucocytes, 11,800; polynuclears, 70 per cent; lymphocytes, 29 per cent; eosinophile, 1; transitionals, 1; small lymphocytes, 26; large lymphocytes, 30. Blood count, May 23, negaitve. Wassermann negative, as were other laboratory findings.

Since leaving the hospital this case has progressed favorably; mastoid wounds have healed.

The interesting features in this case were the occurrence of spontaneous nystagmus, together with symptoms of intracranial complications, and a case of operative double mastoid. There was a history of inter-marriage which the family was loath to disclose and occurred in the second generation.

121 East 60th Street.

BRAIN ABSCESS IN FRONTAL LOBE RESULTING FROM ACUTE FRONTAL SINUSITIS AND OSTEOMYELITIS OF ORBITAL PLATE.*

DR. F. H. BRANDT, Los Angeles.

The facts in this case are that we have an apparent recovery after an almost hopeless struggle, passing through a frontal sinus infection, antrum infection, osteomyelitis, brain abscess and two distinct attacks of meningitis.

Miss G. H., age 17 years, mother living and well. Father and an only sister died of influenza. Her personal history starts with a difficult forceps delivery, marks of which may be seen over left frontal plate. With the exception of whooping cough and measles, her history is negative. In 1925, she struck her forehead while diving, resulting in a swelling between the eyes, of three days' duration. Patient came to my office Sept. 9, 1927, complaining of pain over the right eye. She thought pain was due to having gone swimming two days before; also had a cold two weeks before. Examination showed nothing abnormal about the face. Pressure over right orbital rather relieved than intensified the pain. Tapping on top of head localized the pain in the right frontal sinus region. Nose examination showed a fairly straight septum, turbinates normal size and color, no discharge apparent from either middle meatus. After shrinking, suction was applied, with negative results. Transillumination was also negative. Temperature was 99°, pulse 80. She reported the following day with practically no change excepting a possible slight puffiness over the right eye, temperature 100°. X-ray at this time showed a slight cloudiness in the right frontal sinus, all other sinuses being clear. An intranasal frontal sinus operation was advised, but refused on account of mother's absence from the city. The following day swelling had increased over upper right eyelid, pain was more intense, with a temperature of 102°. The fourth day the eye was closed, temperature 103°, white blood count was 16,100. Mother being on the way home, I insisted on the intranasal operation, which consisted of resection of anterior end of middle turbinate, removing the aggar nasi and opening of the anterior ethmoid cells. This gave free access to the frontal sinus, which was filled with pus and which

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on culture proved to be staphylococcus albus. Drainage was good, temperature decreased from 104° to 101° the next day, and pain was much less. The swelling of the upper eyelid on the third day was no better. Pain became worse and the temperature increased to 102°. On account of these findings I opened up the sinus externally and found pus outside of the bone. Enough bone was removed to have a good view of the sinus, opening into the nose was good, and no other change in the mucous membrane noted. Rubber tissue drain was placed in the sinus. Profuse discharge of pus followed, with a decrease of temperature to about normal in 10 days. The persistent swelling in the nose, however, was very annoying. Suspecting the maxillary sinus, a picture was taken, which showed this dark in contra-distinction to the first X-ray, which had shown no change. Irrigation of the antrum verified the X-ray picture; antrum was filled with pus. This was repeated four times, which gave no apparent result, and a window was made underneath the lower turbinate. This brought down the swelling fairly well, as well as the temperature, and remained normal. The patient left the hospital on the twentieth day. Drainage became less with rubber tube in place. She had, however, more or less headache off and on. On the twenty-sixth day she developed a paresthesia of the left arm. This, of course, was quite a positive sign of trouble in the brain, which was not out of order owing to the persistance of the headache. The retina showed edema in both eyes. Temperature had a tendency to be subnormal. Outside of the paresthesia there was no change in the nervous system at this time. Patient was sent to the General Hospital, white count was made and was: White blood cells, 14,100; poly., 67 per cent; lymph., 23 per cent; mono., 10 per cent.

Spinal fluid was slightly under pressure, cell count 80, mostly monocytes; globulin 0, albumin 0, sugar 0. Dr. Allen saw case in consultation and reported no localized process above frontal sinus. Kernig not definite. No changes in reflexes. Dr. George H. Patterson saw the case the following day and reports: Patient conscious, no frontal headache at present, left eye reacts to L. and A. Some degree of involvement of discs, inferior and nasal quadrant is blurred, some engorgement of veins, suggesting early optic neuritis. Deep reflexes, no apparent change right or left, hyperaction of left biceps. No Kernig or Babinski. Temperature subnormal. Suggests lateral stereo of skull. W.B.C. and spinal puncture.

Dr. Leon Meyers saw the case the following day. *Report:* Patient presents evidence of pyramidal tract irritation on right side. Bilateral choked disc. No pupillary abnormality. No nystagmus, no

ocular palsies. Some ataxia and station weak. Very little cervical rigidity. Abdominal reflexes present. Abscess right frontal lobe.

Impression: Dr. Carters' report on the X-ray is as follows: Drain in right frontal sinus. Right frontal sinus very small, the septum deviates slightly to right. Right ethmoid and antrum also clouded. The septum between the sphenoids also deviates to right. Right sphenoid small and questionably clouded. Additional views of the skull, particularly the right lateral stereo, show a questionable coarse, cancellous structure at the inner table above the right orbit. This is somewhat suggestive, though not conclusive, of an early destructive process in this region. The frontal areas on the whole are thin and the convolutional markings are prominent, which is not conclusive of pathology.

Dr. Patterson's notes on the stereo are as follows: Erosion of posterior wall of right frontal sinus. Eyegrounds show early choking of discs. Reflexes: *1.* Increased left upper extremity. *2.* History of paresthesia left arm. *3.* White count, 14,000. *4.* Increased spinal fluid pressure. *Impression:* Frontal lobe abscess, and suggests exploration for abscess.

With these findings we decided to reopen the sinus wound. The bony opening was enlarged laterally in order to get a better view. The sinus was filled with granulations. Small area of necrosis was found on perpendicular plate between the frontal sinuses. No pus was found in left frontal sinus. Posterior wall was rough. On curetting found a sequestrum, size about ½x1 inch, entirely separated. The dura appeared reddened. The dura was adherent around the opening and was not disturbed. Incision into the dura gave no sign of pus. A ventricular needle was now passed backwards for about 2½ inches, which met no resistance. The next punctures made little more laterally and gave the same result. The third puncture made still more to the side, met a resistance at the distance of 2 inches, and, going through, this was followed by a flow of foul-smelling pus. The next step was to slip a rubber tube over the needle into the abscess cavity. Iodoform drain in the frontal sinus. The amount of pus evacuated was about 2 ounces.

Culture from this pus was also the staphylococcus albus. The patient did not develop a meningitis following this procedure. Headache was gone most of the time. Temperature was normal, choked discs became less. Outside of a little paresthesia in the right arm, she was fairly comfortable. The tube was pushed out little by little

Dr. Meyers saw the case again Nov. 23, 1927, five weeks after the operation, and reports: No evidence of extension of trouble. Left disc still somewhat elevated. Right not examined.

Dec. 11, seven weeks after the operation, the temperature jumped to 102°, pulse 120, with nausea and vomiting and cervical rigidity. Evidence of acute meningitis.

Dr. Meyers again saw the case and reported: Good deal of neck rigidity. Slight Kernig, evidence of pyramidal tract degeneration on left side. Slight tremor of hands. Marked elevation of left disc. No evidence of cranial nerve involvement. *Impression:* Pus over right frontal lobe, acute meningitis. *Advise:* Drainage and spinal

Fig. 1. Shows a slight cloudiness in right frontal sinus. Both antra clear.

puncture. Spinal fluid showed pressure greatly increased. Fluid opalescent. Cells, 3,900; polys., 95 per cent. Globulin increased.

Patient was taken to the operating room. Wound opened. The frontal sinus was filled with granulation tissue and brain substance. A large part of the posterior wall was gone, apparently an extension of the osteomyelitis, and undoubtedly was provocative in causing this meningitis. A ventricular needle was passed up in the region of the abscess to look for closed off abscess, with negative results. Considerable necrotic material was removed from the ethmoid region.

Two rubber tubes were placed in the frontal sinus. Proctoclysis was started, 5 per cent glucose and 2 per cent soda, 1,000 c.c. Repeated in five hours. The following day we had all the symptoms of meningitis, severe pain in back of neck, with nausea and vomiting

occasionally. Dec. 13, another spinal puncture was done, which showed the cell count of 4,139, white blood count, 13,350.

From now on she improved. Dec. 17, the spinal fluid showed only 270 cells and patient did fairly well, when on Dec. 22 she complained of severe pain in back of neck. Temperature went up to 102°. Coincident with this, we had a herniation of brain the size of a walnut. She became totally blind in the left eye and was delirious. Dec. 26, the temperature went up to 104° and she was vomiting. This kept up for several days. At this time we had also a fairly complete facial paralysis on the right side. She was entirely unresponsive at times. On Dec. 30, she started to improve; at this time, outside of the herniation of brain tissue, there was no discharge from the frontal sinus.

She left the hospital Jan. 5, not in the best of condition, but gradually improved. The hernia has gradually decreased, and outside of a little fullness she looks fairly presentable. Feb. 10, she experienced an epileptoid convulsion, which recurred within a week, but not since. As far as I could make out this was of the Jacksonian type. She is in fairly good shape now. Dr. Ray Irvine reports on the eyes the following: Media clear, disc well outlined, little pallor on temporal side. No hemorrhages or exudates. Both eyes show a myopic astigmatism, which in the right eye can almost be brought up to 20/20 vision; in the left, somewhat less. Visual field is slightly reduced.

This case, as you see, has caused a tremendous amount of worry and work, but the results are quite satisfactory. To sum up, we had here a violent infection, producing an osteomyelitis, which I believe by contiguity produced a brain abscess, and later by further extension of the bone process, caused the meningitis. Whether the meningitis was the cause of the facial paralysis I am not prepared to say, which, by the way, has all cleared up. The epileptoid convulsion, mostly in the left extremities, undoubtedly was caused by some process in the right parietal region. Supplementary X-ray report by Dr. R. A. Carter.

Comparison of views of June 19, 1928, with previous views with particular reference to the displacement of the right orbital walls, this appears to have occurred almost completely between the previous examination of Dec. 7, 1927, and the present examination of June 19, 1928. On review there is also evident an area of absent bone, with smooth edges at the roof of the right orbit, not giving appearance of a present active destructive lesion of bone.

Dr. Seymour, Chairman of Staff: This paper and the description of this case would certainly lead us to believe that all things are possible in medicine and surgery, however desperate the case may be. It is exceedingly interesting and I would like to hear some of the discussion of this case. Anyone else discuss this paper, any constructive criticism? Do not be afraid of hurting Dr. Brandt's feelings if you do not agree with him. I am sure that he would be delighted that you had the courage of your own convictions.

Dr. Patterson: In the discussion of this case there are one or two things I would like to say. In the first place, this person had a

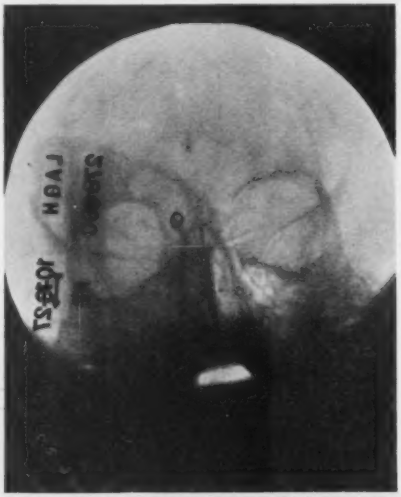

Fig. 2. Shows tube in frontal sinus, right antrum dark.

fungus which was extending out through the frontal sinus. There is always a tendency to remove these brain hernias by surgery. However, it is our experience that when the intracranial pressure is reduced, these usually take care of themselves, and, as you can see in this case, such a condition has taken place here. There was an erosion of the posterior wall of the frontal sinus in this case, as shown by X-ray, and the abscess was approached and drained through this area. I am forced to disagree with this method of approach in operating for an abscess of the brain.

We believe in our department that the clean method of approach is the most logical one. In the first place, if an approach is made through an infected area, such as a mastoid or frontal sinus, and the

abscess is not found, infected material is carried through the brain, and the danger of meningitis is encountered. In the second place, brain abscesses which become chronic are sometimes sterile when cultured and it would seem that a clean approach to such a condition would be the one of choice.

Dr. Meyers: I would like to say a word about the diagnosis of this case. When I examined the girl, she showed a frontal sinus infection, papillodema, and a flexion reflex in the left leg, and it was on the basis of these findings that I assumed the presence of a frontal lobe abscess in this case. I published a paper in 1923, under the title of "The Progression Reflex", in which I showed that in the early cases of pyramidal tract irritation, passive flexion of the big toe

Fig. 3. Shows some change in posterior wall of orbit.

produces flexion in all the articulations of the leg, and that the leg remains in extension if the other leg is, during this procedure, fully flexed. The girl, as you have noticed in the report of the case, shows practically no deviation in the reflexes. She had no Babinski, and the only thing suggestive of involvement of the pyramidal tract to the left leg was the presence of the flexion reflex in that leg. In view of the fact that she also had a choking of the discs, which was more marked on the right side, I thought it was almost certain that she had a frontal abscess on that side. I was much pleased to hear afterwards that Dr. Brandt operated on this case, and the diagnosis was found to have been correct.

The relatively small number of cases reported in literature leads me to believe that the occurrence of these cases is rather infrequent. Taking into consideration the prevalence of nasal sinus infections, it is somewhat difficult to account for the small number of cases involved.

Dr. Chas. J. Imperatori[1] reports five cases of frontal lobe abscess, all cases never below 28, two of the five occurring in girls. Four had an external operation, one an intranasal and later an external operation. His deductions of apparent method of invasion are:

Case 1: Direct extension from frontal sinus; lived. *Case 2: By* metastatic infection; lived. *Case 3:* By direct extension; lived 14

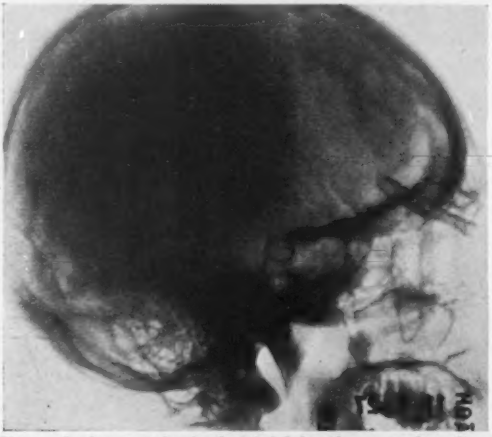

Fig. 4. Shows drainage tube in frontal lobe three weeks after operation. Tube shortened about one-half inch during this time.

months. *Case 4:* By direct extension; died. *Case 5:* By direct extension and osteomyelitis; died.

Summary of Symptoms: Case 1: Headache especially at night at sight of lesion. *Case 2:* Occasional vomiting. *Case 3:* Chemosis and some eyeground changes. *Case 4:* Optic neuritis when abscess localized. *Case 5:* High temperature toward A. M., pulse in relation. *Case 6:* Convulsions.

Frithy of Leegard[2] reports four cases and reviews literature in 87 cases. Most frequent symptoms in headache. Usually some paralysis in arm and leg. In 49 cases, optic neuritis.

F. P. Sanders and Garnet Halloran[3]: Right frontal lobe abscess in a man age 21 years; left facial paralysis, abducens paralysis, choked disc, X-ray negative. Head deviated to right as well as right eye. Flaccidity of muscles of left arm and face.

Aloin[4] reports an abscess of left frontal lobe following chronic frontal sinusitis.

Berens[5] reports case of frontal lobe abscess following chronic frontal sinusitis.

Bryan[6] reports a case of frontal lobe abscess in a boy age 15 years. Infection apparently by direct extension from frontal sinus.

Fig. 5. Shows large defect of posterior wall of orbit as well as part of the ethmoid cells. All sinuses are relatively clear. Window in left antrum can be made out.

Skillern[7] reported a frontal lobe abscess following acute ethmoiditis in a young woman.

Swain[8] describes a frontal lobe abscess occurring in a patient with frontal sinusitis.

W. F. Calpas[9] reports three cases frontal lobe abscess, complication of the cranial bones, all recovered.

E. M. Seydell[10] describes a case of frontal sinusitis complicated by frontal lobe abscess.

D. A. Vanderhoof[11] reports a case, man, age 21 years, acute frontal sinus infection. Intranasal operation done at first, later external. Eye changes marked, proptosis, corneal anesthesia. Retinal conges-

tion. Postmortem showed: Frontal sinusitis with external perforation, extra and subdural abscess, abscess of right cavernous sinus.

Sam Salinger[12] reports a case, man, age 18 years, frontal lobe abscess following frontal ethmoid sinusitis. Left hospital as cured, returned two weeks later with severe headache, nausea and vomiting. Bilateral neuroretinitis, some swelling of nerve head. Wound was reopened, no pus found. Brain explored to depth of 4 c.m. in all directions, no pus found. Died in 24 hours. Autopsy: Abscess size of hazel nut found encapsulated, back of the original abscess—no communication found.

J. Mackinzie Brown[13] reports two cases. Male, age 19 years, swelling over left frontal sinus and upper part of sternum, severe headache, left optic disc swollen, W.B.C., 17,300. Vomiting, unconscious, left pupil dilated, no response to L. & A. External frontal sinus operation. Posterior wall necrotic, removed and pus outside of dura, no sinus into dura. A little later symptoms called for a second operation; an ounce of pus was found on temporal side, ½-inch in frontal lobe; recovery. The second case, male, age 50 years. Had an external frontal sinus operation, posterior wall was necrosed, granulation on dura, apparent recovery. Two months later became suddenly ill and died. Postmortem: Frontal lobe abscess.

G. Berry[14] reports a case in a woman, age 24 years, of acute left frontoethmoiditis apparently recovered, when 3½ weeks later experienced epileptiform convulsions, followed by headache, nausea and vomiting. Pulse 66, normal temperature, left exophthalmos, suggestive left optic neuritis, paralysis of left right rectus. Intranasal surgery was done and frontal lobe abscess evacuated through posterior ethmoidal roof, proven by X-ray. Patient went home after four months, apparently cured; convulsions same night with vomiting; repeated off and on for about nine months. Then for three years was free of these seizures, when she developed meningitis and died. Postmortem showed a large frontal lobe abscess similar to one found four years previously. Meningitis resulted from rupture of the base of the abscess.

H. H. Kerr[15] reports six cases of frontal lobe abscesses. Man, age 25 years, frontal sinus and antrum infection, causing an abscess in frontal lobe, abscess ruptured into ventricle. Bilateral subtemporal decompression; died. Boy, age 16 years, cerebrospinal rhinorrhea. Pneumococcus, craniotomy; recovered. Man, age 29 years, gunshot wound of frontal lobe, staphylococcus, craniotomy; recovered. Man, age 17 years, ethmoiditis, staphylococcus infection, craniotomy; recovered. Girl, age 10 years, nasopharyngeal infection, craniotomy;

died. Woman, age 28 years, frontal sinusitis and osteomyelitis of frontal bone. Exploratory craniotomy; died. Kerr writes: Abscess of frontal lobe has been recognized only about one-half of reported cases.

Walter Lillie[16] reports one case. Boy, age 14 years, eight months previously had cold, with edema of left eyelid. This cleared up, but later resulted in a frontal lobe abscess, optic neuritis.

In conclusion, we might say:

1. Most cases found seem to favor the young, the diploe being more spacious and the mucosa being in more intimate relation with the bone tissue, and the lymphatic and vascular system being more profuse, might account in a way for the occurrence in early life.

2. Headache which is persistent after thorough drainage for a frontal sinus operation should always be watched very closely.

3. The occurrence of paresthesia of the opposite arm almost always means a frontal lobe involvement.

4. Eyeground changes should be watched very closely; when present is a sure index of increased brain pressure, and a timely eradication of this pressure will obviate changes in the optic nerve.

5. Drainage in brain abscess should rather be prolonged than shortened.

6. Osteomyelitis of the cranial bone has a tendency to be progressive, which in our case accounted for two distinct attacks of meningitis.

BIBLIOGRAPHY.

1. IMPERATORI, CHAS. J.: *Med. Jour. and Rec.*, Vol. CXXXVI, Oct. 5, 1927.
2. FRITHY: Annals of Otol., Rhinol. and Laryngol., p. 28, 1919.
3. SANDERS, F. P., and HALLORAN, GARNET: *Med. Jour. of Australia*, Aug. 27, 1927.
4. ALOIN: Lyon Med., 104, Aug. 27, 1927.
5. BERENS: Trans. Amer. Laryngol. Assoc., 1920, p. 156.
6. BRYAN: Ibid., 1921.
7. SKILLERN: Ibid., 1922, p. 56.
8. SWAIN: Ibid., 1926, p. 56.
9. CALPAS, W. F.: Laryngol., Rhinol. and Otol. Soc., 1926, p. 455.
10. SEYDELL, E. M.: Ibid., 1926, p. 449.
11. VANDERHOFF, D. A.: Annals of Otol., Rhinol. and Laryngol., Vol. XXXV, p. 508.
12. SALINGER, SAM: Annals of Otol., Rhinol. and Laryngol., Vol. XXXIV, p. 210.
13. BROWN, M. F.: Annals of Otol., Rhinol. and Laryngol., Vol. XXXVI, No. 3.
14. BERRY, G.: THE LARYNGOSCOPE, Vol. XXXIV, No. 5, 1924.
15. KERR, H. H.: Arch. of Surgery, Vol. 6, 1923, p. 297.
16. LILLIE, W.: *Jour. A. M. A.*, Vol. 89, No. 25.

1052 W. Sixth Street.

THE REASONS FOR AND RESULTS OF A NEW OPERATION FOR DEAFNESS.

DR. J. NORVAL WATT, Durban, Natal, S. Africa.

The inaccessibility and difficulty of medication of the Eustachian tube has rendered the treatment of deafness far from satisfactory. The majority of cases of deafness can be traced to such causes as catarrh, or actual narrowing of the tube, dependent primarily on other sites of trouble, *e. g.*, nasal and sinus conditions, tonsils and adenoids, etc. Clearing up of the primary condition does not seem to affect the deafness to any extent, because the tube, except in a few cases, still remains closed. Inflation, catheterization, politzerization, etc., have either a very transient effect, none at all, or tend to drive matter already in the tube or carried by the catheter, etc., from the nose into the middle ear. Tubal obstruction is one of the most unsatisfactory conditions to treat with which the specialist comes in contact.

On the patency of the Eustachian tube depends the mobility of the ossicular chain, on which depends the free excursions of the two membranes, the tympanum and the membrane of the fenestra ovalis. A negative pressure in the middle ear, and a positive, or atmospheric pressure, on the external aspect of the drum, causes a marked retraction of the drum and a bulging of the membrane into the fenestra ovalis. The ossicular joints are jammed together and so there is no movement and the sound vibrations are not conveyed to the inner ear via the drum and the ossicular chain.

The result of this state of affairs is intractable noises in the head, tinnitus and deafness. In temporary cases the deafness never seems to completely resolve; each attack making the patient "harder of hearing" until the deafness becomes chronic. The incessant head noises seem to be worse at night and in many cases insomnia is the forerunner of neurasthenia and mental disorder. In a great many cases the noises are incessant and it is for this reason the patient consults the specialist.

The first case operated on by the writer is of interest from the viewpoint of showing the rapidity of the result.

Patient came to me for deafness and noises in the head. He had chronic otitis media in the left ear and only heard by bone conduction.

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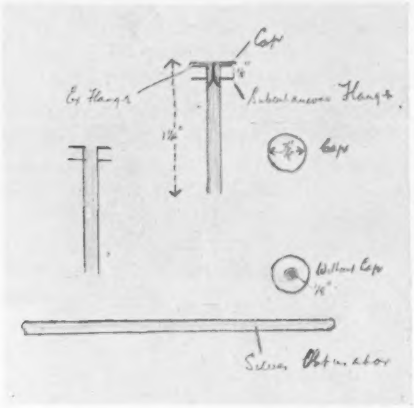
This was of many years' standing. Incessant noises in both sides of head. Right ear for the last three years had gradually been getting deafer, and for the last six months could hear practically nothing by air conduction. He could occasionally manage to pick up a few words by watching the lips and if "bawled at". The drum was markedly retracted, had lost its lustre, and could be moved by suction. Both Eustachian tubes were closed and could not be inflated. Marked nasal obstruction, tonsils and adenoids present; probably the original cause of the left chronic otitis media.

Operation on right ear; 24 hours later removed dressing to remove any blood accumulation. Patient heard perfectly well, wristlet watch



at 9 inches, whispered voice, ordinary conversation; writer was told "not to speak so loudly"; noises had completely disappeared and have never returned. Tinnitus still present in the left ear. Four days after did a submucous resection of septum and removed tonsils and adenoids with a view to drying up the left chronic otitis media and enabling patient to breathe freely. Patient went home eight days after admission, hearing perfectly well. Noises still present in left ear. Were this a solitary case one might say that the nasal and throat operation would have been all that was necessary, but it is not and, as before mentioned, the writer's experiences of nasal and throat operations for deafness have not been so satisfactory as to enable him to be very optimistic to patients. In the writer's opinion, before labeling cases of deafness as incurable, the operation as out-

lined below should be tried. No harm has resulted in the cases so far and the results have been remarkable. As far as the writer is aware there is no literature or publication dealing with this method of elimination of the Eustachian tube. Below will be found a sketch and description with the measurements of the tube made to the writer's order.

The Operation: Incision as for opening the mastoid antrum down to bone. Periosteum reflected in both directions. Antrum is opened and the aditus exposed. Opening of aditus made larger by chipping pieces off all walls except the floor. A small sound is introduced into aditus and its patency demonstrated. All chips of bone are removed and the antral cavity swabbed out and dried. The tube is now slightly curved over an obturator so that the upper or external flange will be flat on the skin surface. Distance from the skin surface to the enlarged antral end of the aditus is measured off on the tube from the external flange and the surplus is removed by a fret saw and the end filed smooth. The tube is now put in position after being sterilized and the skin brought together between the two flanges and completely sewn up. It is advisable to leave the cap off the tube for a day or two to enable any serious discharge which may present itself from the blood to drain away. Stitches removed on the fourth or fifth day. With aseptic precautions there are no complications. The cap of the tube can be removed and replaced when desired.

The tube is made of: 24 ct. gold as it is easiest of manipulation. Tube in one piece and turned so that there is no solder, and it is 1¼ inches long so as to allow for cutting. Distance between flanges, ⅛-inch. Width of flanges from edge of tube, an infinitesimal bit smaller than ⅛-inch. Cap slightly larger than upper flange and flat, and works like the top of a collar stud.

A solid silver obturator slightly smaller in diameter than the tube is used. The tube is bent on this obturator to avoid kinking.

14 Anstey's Buildings.

MENIERE'S DISEASE. REPORT OF A CASE.

DR. LAWRENCE K. GUNDRUM, Los Angeles.

In 1848, Méniére published the following note: "I have seen a young girl stricken with complete deafness, absolute in the short space of a few hours. Traveling in an uncovered vehicle, she was exposed during the night to a very severe cold at the time of her periods and the hearing was lost without the ears being the seat of pain. Death, which promptly followed, permitted me to dissect carefully the two temporals and I found in the whole labyrinth a sort of reddish plastic lymph, which seemed the product of an exudation of all membranous surfaces of the internal ear. In a similar case, but one which did not end in death until two months later, I found the same condition." Thirteen years later he described the same case and stated that vertigo, tinnitus, nausea and vomiting were present. Whether the case Méniére described was as he thought or a meningitis can never be proven.

However, since Méniére's description more than 300 monographs have been written on this subject. Many writers have described any condition resembling this as Méniére's disease. Thus we have described "Méniére's Symptoms", "Méniére's Symptom Complex", "True Méniére's Disease", and "Apoplectic Méniére's Disease". One author[1] goes so far as to classify it under the general heading of "Migrain". Politzer[2] distinguishes between the "Apoplectic form of Méniére's disease" and "Méniére's Symptoms". He describes the apoplectic form as having a sudden onset during good health, while Méniére's symptoms may come on during the course of acute or chronic affections of the ear or lesions of the central nervous system or any toxic condition affecting the inner ear.

A variety of causes have been given as factors in producing this condition. Among these are heat stroke, pernicious anemia, nephritis, syphilis, hypertention and the leukemias. The patients are usually in middle life. The onset is sudden. The patient usually falls to the affected side. Vertigo, tinnitus, nausea and vomiting are always present. Consciousness may or may not be lost. If lost, it is regained in a short time. Bull believes that the loss of consciousness is due to mental depression. Examination usually shows normal eardrums, with hearing either partially or completely destroyed. The

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symptoms may persist for a few hours, or for a period of months. Usually all disappear except the deafness, which is permanent. No treatment up to the present time has had any influence on the condition.

Very few postmortem examinations have been made, due to the fact that the disease is not often fatal and the inner ear is so often neglected in routine autopsies. Hemorrhage into the labyrinth has been found in a number of cases and is generally believed to be the cause of the symptoms. Parry[3] believes that they are caused not only by increased pressure, but also by changed quality of the lymph.

Case Report: Joseph S. came under observation at the Los Angeles General Hospital, Dec. 15, 1927. *Chief complaint:* Dizziness, deafness and ringing in the right ear.

Family history: Patient remembers nothing of parents, brothers or sisters, as he spent his early life in an orphanage.

Past history: Had the usual childhood diseases. Acute gonorrhea in 1913, with urethral discharge, which persisted six weeks. Married in 1923, divorced in 1925. Acquired gonorrhea again in May, 1927. This attack seemed to be much more severe than the previous one. Was treated by his family physician, who gave frequent urethral injections and "serum treatments" by injections into the arm. The last intramuscular injection was given June 27, 1927, at 7 a. m.

Present illness: June 27, 1927, at about 5 p. m., while eating dinner the patient suddenly became nauseated and lost consciousness. He thinks he remained unconscious for about 20 minutes. Was taken immediately to a private hospital. When consciousness returned the patient was nauseated and vomited continually. This continued for about 48 hours. About this time the patient noticed he was quite deaf in the right ear and tinnitus was present. He apparently had true vertigo, as he stated that "articles in the room seemed to move about constantly". The vertigo was increased by movement. He remained in bed until July 11, when he attempted to get up. He found he was unable to do this without intense nausea and vertigo. July 14, he got out of bed and tried to walk, but found he was unable to do this, as he would stagger from side to side, and was more liable to fall to the right. At the time of our first examination, Dec. 15, 1927, he was considerably improved, although he was still unable to walk without assistance. He was admitted to the General Hospital, Oct. 27, 1927. Examination at this time showed temperature 98° F., pulse 76, respiration 20, height 5 feet 5½ inches, weight 132 pounds. Blood pressure: systolic 115 m., diastolic 70 m. Head: pupils react to light and accommodation. Chest: scar of stab over

right clavicle. Lungs, heart and abdomen normal. Extremities: some atrophy of muscles of right arm due to an old injury of the brachial plexus. Reflexes normal. Spinal fluid: clear under normal pressure, sugar content normal, globulin test negative, cells 4 per c.m.m. Sterile after 48 hours' incubation. Wassermann negative. Blood Wassermann, Oct. 21, Dec. 2, Dec. 14 (provocative): all negative. Complete blood examination, Nov. 22, normal. Urine, Oct. 27, normal. X-ray, Dec. 1, showed no periapical pathology; paranasal sinuses: frontals small, clear, shallow; ethmoids clear, maxillaries medium size, clear in upper portions, pathology not ruled out in lower portions; sphenoids clear. Stereo of chest negative. Dental examination showed no pathology in teeth.

Ear, nose and throat examination: Ears: Both membrana tympani normal. Examination of the vestibular apparatus showed: No after-turning nystagmus, motion sensing or past pointing from right ear, no reactions after douching 3 minutes, 30 seconds. Left, normal responses to the turnings and caloric tests. Measurements of hearing with the audio amplifier showed an average tonal hearing of about 5 per cent in the right ear and 77.7 per cent in the left. The upper limit in the right ear was 512 d.v. per second. In the left, 20,000 d.v. per second. The impairment in the left ear was conductive in type. Nose normal. Both maxillary sinuses were irrigated, with negative results. Throat normal. Tonsils had been removed clean. Larynx: some lagging of left cord. Arytenoids freely movable.

Re-examination, April 12, 1928, all of the vestibular symptoms had disappeared, was able to walk without assistance. However, the deafness was still present. Vestibular tests were the same as on the previous examination.

Re-examination Aug. 14, 1928. Deafness was still present. Responses to turnings were the same on both sides, but about 60 per cent normal. However, douching the right ear gave no responses in three minutes. Douching the left ear gave normal responses. Larynx exactly the same as on previous examination.

Comment: No history could be elicited of any laryngeal symptoms at the time of the attack. Together with the fact that the condition has remained unchanged for more than a year makes it seem probable that this condition was one of long standing and was in no way associated with the attack of Méniére's disease. This case seems to be one of true Méniére's disease. The following classical symptoms were present:

1. The sudden onset.

2. The symptoms of irritation of the vestibular apparatus, *i. e.*, nausea and vomiting, vertigo and staggering gait.

3. The cochlear symptoms characterized by tinnitus and the sudden loss of hearing on the affected side.

The cause after exhaustive study could not be determined. The patient believes that the substance injected in his arm caused the attack. However, this seems improbable.

BIBLIOGRAPHY.

1. GOULD, GEORGE M.: The Myth and Mystery of Meniere's Disease. Med. Record, Vol. 74, No. 16, p. 745, Oct. 31, 1908.
2. POLITZER, ADAM: A Textbook of the Diseases of the Ear. 732-35. Lea Bros. & Co., 1903.
3. PARRY, T. WILSON: Annals of Oto., Rhin. and Laryn. Vol. XVI, No. 3, p. 558, Sept., 1905.

1920 Wilshire Boulevard.

HERPES ZOSTER (INTERCOSTAL) AND INTERCOSTAL NEURALGIA RELIEVED BY SPHENO-PALATINE GANGLION TREATMENT.

DR. SIMON L. RUSKIN, New York.

Case 1: Mrs. D. C., age 32 years, was referred to me by Dr. Harry Handelman with a history of having had for the last three weeks pain along the course of the ninth left intercostal nerve. This was followed by a typical eruption of herpetic vesicles and the pain became lancinating in character and associated with troublesome itching. In addition, she suffered from general nervousness, a choking sensation in the throat, watery nasal discharge, and a sensation of tightness across the bridge of the nose. She has had the choky sensation in the throat for a few months, and within the same period she noted that her voice had become low-pitched. She also suffered a sensation of fullness in her left ear.

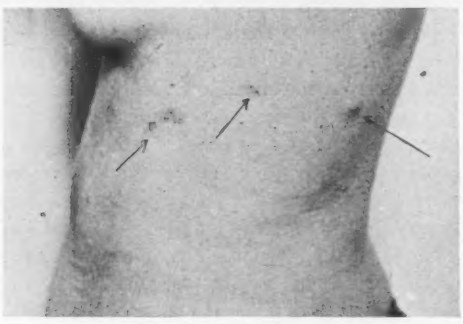
Nasal examination revealed a slight atrophic rhinitis. Her tonsils were deeply buried and diseased. Larynx was negative.

Examination of the abdomen revealed a typical herpes zoster eruption, as in the accompanying illustration.

Cocainization of the nasal ganglion gave immediate relief from the zoster pains. That night she had her first comfortable rest since the onset of her attack. She received daily nasal ganglion applications for the next four days, with almost complete relief. Simultaneous with the relief of the zoster manifestations came the relief of the throat and nasal disturbances.

It would seem like a far cry to correlate herpes zoster affecting the spinal root ganglion of the ninth intercostal nerve with the sensory connection. It is, however, probable that the pathway lies along the sympathetic fibres passing from the nasal ganglion to the cervical sympathetic ganglion, and from there to the sympathetic ganglion communicating with the ninth spinal ganglion.

The correlated findings in the nose and throat are supported by the interesting observations of Leriche and Fontaine, who noted cer-

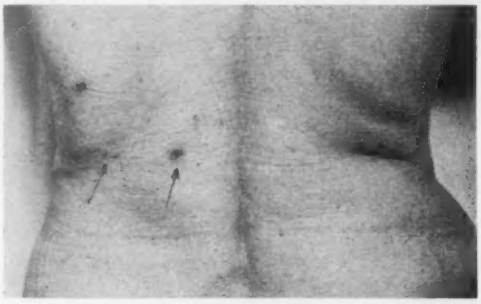


tain morbid phenomena in the throat after an operation upon the cervical sympathetic; dryness of the throat, hoarseness and difficulty in swallowing; associated with hyperemia and edema of the tongue, larynx and pharynx. In a recent case in which the middle cervical ganglion only was removed, all the symptoms were as marked as after more extensive operations. They suggest that this ganglion may represent a vasomotor centre for the larynx.

In my paper, "Headaches and Systemic Disturbances of Nasal Ganglion Origin", I indicated the close relationship between vaso-

motor disturbances and sensory pain particularly in the musculature. The relief of pain in angina pectoris by section of the cervical sympathetic points in the same direction. Heitger relieved the pain of angina pectoris by cocainization of the nasal ganglion. Sluder feels that this is unquestionably the blocking of the transmission pathway of sympathetic nerve pain.

Case 2: Mrs. F., age 40 years, was stricken suddenly with a severe pain in the left side of the chest. The pain was so severe that the



patient could hardly be moved. Dr. Barnet Joseph examined her during the attack and diagnosed her condition as interco tal neuralgia.

Blocking the nasal ganglion gave her immediate relief. The neuralgia did not recur. It is interesting to note that the previous year she had suffered from a troublesome sciatica, which cleared after the removal of her diseased tonsils.

BIBLIOGRAPHY.

SLUDER, GREENFIELD: Nasal Neurology, Headaches and Eye Disorders. C. V. Mosby Co., 1927, pp. 274-278.

LERICHE, R., and FONTAINE, R.: Physiologic Role of Middle Cervical Ganglion. Also abstract, *Jour. A. M. A.*, Nov. 13, 1926, LXXXVII, 1689.

HEITGER, JOSEPH D.: A Consideration of the Sphenoid and Posterior Ethmoid Paranasal Cells as an Etiologic Factor in So-called Brachial Neuritis. *South. Med. Jour.*, March, 1925, XVIII, No. 3, pp. 218-221.

RUSKIN, SIMON L.: The Sensory Field of the Facial Nerve. Archives of Oto-Laryngol., April, 1928, Vol. 7, pp. 351-358.

RUSKIN, SIMON L.: Herpes Zoster Oticus Relieved by Sphenopalatine Ganglion Treatment. THE LARYNGOSCOPE, April, 1925.

RUSKIN, SIMON L.: Contributions to the Study of the Sphenopalatine Ganglion. THE LARYNGOSCOPE, Feb., 1925.

RUSKIN, SIMON L.: The Nasal (Sphenopalatine) Ganglion, In Relation to Itching in the Auditory Canal and to Tinnitus Aurium. Archives of Oto-Laryngol., Sept., 1925, Vol. 2, pp. 269 and 270.

RUSKIN, SIMON L.: Tic Doloureux and Trigeminal Neuralgia, Relieved by Treatment of the Nasal (Sphenopalatine) Ganglion. Archives of Oto-Laryngol., Dec., 1925, Vol. 2, pp. 584-586.

351 West 86th Street.

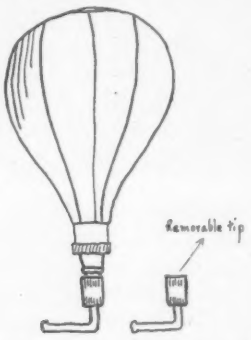
A NEW NASAL DOUCHE AND AURAL IRRIGATOR.*

DR. ROBERT M. COLBERT, New York.

The inadequate means at hand for nasal irrigation has prompted me to design this little apparatus which I have found most helpful.

The usefulness of nasal irrigation has been frequently stressed by prominent rhinologists. Men like J. Solis Cohen, DeSchweinitz and Ballenger have advocated it often.

Many douches and irrigators have been devised, but a combination of an efficient irrigator and one that is practical by its simplicity has not come under my observation.



Most all nasal douches are constructed to occlude the nares. Since the nasal passages are usually blocked when a douche is to be used, such occlusion is wrought with serious danger, as the considerable pressure developed causes either a washing into or an increased pneumatic pressure into the Eustachian orifices with a frequent middle ear infection resulting therefrom.

In this apparatus this detriment has been to a great extent obviated. The insertion of a small calibre rigid catheter into the nose allows ample space around it for the dissipation of excess pressure and a true flushing is accomplished. The fluid can be passed under regulated pressure into one nasal cavity, flowing through the nasopharynx and out through the other nostril, as well as around the inserted catheter. Besides, the construction is so arranged that by an angular

*Read at the New York Academy of Medicine, May 4, 1927, before the Harlem Medical Society.

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connecting piece the bulb is held above the site of return flow and thereby avoids the usual mess and wetting which is so frequently experienced in nasal douching. The fluid runs directly out of the nose into a basin held under the chin.

A longer catheter, for the use of the physician only, can be passed beyond any obstruction and thereby flush the posterior nares.

The catheter is quickly detached from the bulb by a slight twist and so allows a rapid refilling of the bulb.

Although the douching alone does not act as a cure for such conditions as sinusitis, chronic catarrhal inflammations, ozena and the like, its action produces a clean surface, upon which an adequate medication may be applied, and at the same time facilitates drainage.

On the other hand, its prophylactic action can hardly be ignored. Under present city conditions, with various air pollutions, it is most advisable to cleanse the air passages of any accumulation that has occurred during the day. With this simple procedure it is a most acceptable function and, in fact, should become part of the nightly toilet and would hardly take as much time as it does to brush the teeth.

There is another use for which this apparatus may be employed, namely, for aural irrigation. The ease and convenience with which the ear canal may be flushed and cerumen removed, without splashing, recommends it as a most desirable means for this purpose.

160 West 87th Street.

RESECTOR FOR REMOVING SHAPED PIECE OF RIB CARTILAGE FOR NASAL TRANSPLANT.*

DR. CARL H. FORNELL, New York.

This instrument consists of an oval-shaped cutting edge at each end of a handle, which is approximately 8 inches in length. One of these ovals is larger than the other, in order to vary the size of cartilage graft as desired. The oval-shaped cutting surface was chosen since the costal cartilages are oval in shape on cross-section, and it was thought that the removal of an oval-shaped graft would leave a

*Read before the New York Academy of Medicine, Section on Laryngology and Rhinology, March 28, 1928.

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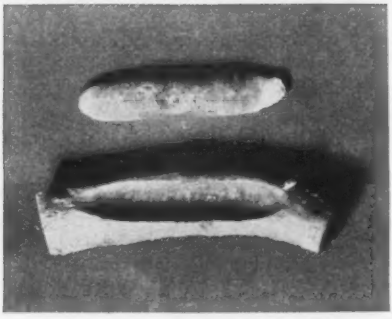
stronger remnant of costal cartilage than if a square or triangular cutting edge were used. A moderate amount of pressure serves to engage the cutting edge in the cartilage and then by a pulling motion the graft can be made as long as the length of the costal cartilage will allow. The use of a scalpel in removing a cartilage graft is open to the criticism that the scalpel may slip, due to the respiratory motions of the patient's chest, thereby causing possible injury to the underlying structures. Removal of the graft by a scalpel is also time-consuming and the resulting graft is likely to be more irregular in shape than that obtained by the use of this instrument. Obtaining



a cartilage graft by the use of a gouge is open to the criticism that the size and shape of the graft is less easily controlled, and there is also the danger of the gouge slipping. The use of the gouge also requires a pushing motion, which is less easy to control than the pulling motion required in the use of this instrument. The safety of this new instrument depends on the fact that the dull portion of the cutting oval slides along the anterior surface of the cartilage and the cutting edge cannot be admitted deeper than the size of the oval will allow.

65 Park Avenue.

THE NEW YORK ACADEMY OF MEDICINE.

SECTION ON LARYNGOLOGY AND RHINOLOGY, IN CONJUNCTION WITH SECTION ON OTOLOGY.

Meeting of April 25, 1928.

Laboratory Aids in Otology. Dr. S. J. Kopetzky.

(Published in THE LARYNGOSCOPE, *June, 1928.)*

DISCUSSION.

DR. T. J. HARRIS: I wish to congratulate Dr. Kopetzky on this admirable paper. You will all agree with me that he has given us a paper that for careful preparation and for practical value is one we rarely have opportunity of hearing. I have followed it with interest and with no little profit. Any discussion must be limited, and it is impossible to cover in a few minutes all the various points that Dr. Kopetzky has touched upon. Perhaps the last thing he said was one of the most important, namely, that after all laboratory aids have been made use of we must depend upon bedside observation. As he spoke, I thought of Dr. Gruening. Many of you did not have opportunity to know him, but many of us did. He was perhaps as scientific in his methods as any man in otology, and he always used to say that all laboratory aids sink into insignificance compared with the ability to do proper bedside work. Dr. Kopetzky's paper is made doubly important by the fact that it is based on his own personal observations. I wonder if you realize that he spoke of 18 cases of sinus thrombosis? He is speaking of his personal experience, and I do not think that many of us make use of many of the things he referred to: I do not think that many of us realize the value of the red blood count as he has stressed it—how important it is to know the rising and falling of the red blood cells. Take on with what we are doing with the repeated examinations of the white cells, it is, as he pointed out, of exceeding value.

Two other things he pointed out we should make more use of, namely, the McKernon resistance chart which, although it dates back many years, is not yet in general use so far as I know. Von Schilling staff cells test is unknown to me, but has proved of exceeding value in Dr. Kopetzky's hands in determining the progress of a case.

In regard to the X-raying of the ear, I would like to say that every word Dr. Kopetzky uttered tonight in regard to the proper appreciation and recognition of its value is true. In a little group of men the other night we had this question up for discussion, and some of the men said they thought that the X-ray as used is of very little value; and one man said—and most any man's opinion in the city did he lay any weight on it—and that because this man was clinically able to follow his case and arrive at his opinion both by the X-ray and also by his own clinical knowledge of the case. We must get away from making our diagnoses purely by the X-ray findings.

In regard to spinal puncture, I would refer to the warning that Dr. Eagleton has given repeatedly. Dr. Kopetzky has spoken of its value and that he is using it in every case. Dr. Eagleton has said there is danger, particularly where there is the possibility of a subarachnoid involvement. Dr. Kopetzky did not refer to leukoplenia. I had a case of sinus thrombosis where we regarded leukoplenia as the outstanding diagnostic feature of the case. It is in regard to the non-suppurative conditions that I was particularly interested in what Dr. Kopetzky had to say about his findings, namely, the classification he makes in regard to progressive and non-progressive deafness.

Once more I wish to congratulate the essayist on a paper that will be of exceedingly value to us all.

DR. R. OTTENBERG: Dr. Kopetzky has laid before us a vast amount of material. His stress on the hemoglobin and red blood count is of importance. It is a thing we often forget. As the result of following many cases, I agree that the steady downward trend is indicative that the infection (mostly with streptococcus) is progressing. The hemolytic streptococcus does destroy blood. As a practical matter, that is very important. It is one of the clinical things that constitute the total picture of the case. I agree with Dr. Kopetzky, as a medical man, that the clinical picture is more important than any laboratory test. No laboratory test will make the diagnosis. One has to put all the parts together to form the clinical picture.

I was not aware that the Von Schilling test was used in otology. I can well see that after careful statistical studies have been made one may be able to set up certain standards which will enable him to say whether or not a case is going on to a serious condition and needs an operation. I have again and again seen otologists differ decidedly as to whether a given mastoid needed operation, and a test of this kind might give very valuable information.

The thing that interested me most was the discussion on blood culture. I differ with Dr. Kopetzky on a few points. He feels that the value of blood cultures has been greatly over-estimated. I cannot help feeling that it cannot be over-estimated; a blood culture is a definite, positive fact. One must know what the report means when one gets it. This subject, the interpretation of positive and negative blood cultures, was discussed very elaborately some years ago by Dr. Libman; what I know is largely learned from that source. One can get a great deal of help, but if he uses them automatically one will be wrong as often as right. For instance, pneumococci in the blood does not mean sinus thrombosis. Blood cultures taken within 24 to 48 hours after operation mean nothing, because the blood is often transiently affected as a result of operation itself. In general, a positive blood culture with streptococcus or pneumoccocus Type III in the presence of mastoiditis points toward sinus thrombosis, provided one can exclude other possible causes of blood infection, such as endocarditis or pneumonia. On the other hand, a negative blood culture never excludes sinus thrombosis because, as Dr. Kopetzky truly said, a thrombosis may sterilize itself and no longer give off bacteria to the bloodstream.

In helping exclude other sources of infection in the bloodstream, the diffential blood culture which I described before this Society last year, is often of help. If one finds a very much larger number of bacteria in one jugular vein than in the other, one can exclude distant foci, such as lungs or heart valves. On the other hand, it must be understood that such a finding does not tell us at all on which side the sinus thrombosis lies.

I wish to call attention to one new laboratory aid which Dr. Kopetzky omitted —you will find it discussed by Dr. Friesner and Dr. Rosen in the *Journal of the American Medical Association*, April 16, 1927—the determination of the amount of calcium in the pus from the ear is a guide in determining the breaking down of the bone in mastoiditis; this is a simple, direct test and properly controlled and, interpreted, should become one of great usefulness.

DR. J. R. LOSEE: I am quite sure that we all appreciate the very masterly manner in which Dr. Kopetzky has treated the subject for discussion this evening. Up to this time few of us have paid much attention to the red blood cell count and hemoglobin estimation in theses cases; and since my attention has been directed to it I have looked up the records in the last 15 cases and have observed a diminishing red cell count with an increase leukocytosis in most of the cases. I beg to suggest, however, that it is well in making these counts to have the same person perform them daily, in order that the relative portion of error will be the same. Although I have had no actual experience with the Von Schilling method of counting polynuclear leukocytes, it seems to me that it is even an earlier indication in determining the extension of the infection than the polynuclear count alone.

I quite agree with the author that every diagnostic and surgical means should be taken to prevent the appearance of organisms in the blood. Once these organisms appear in the bloodstream we may be able to control them by ligation of the vein, but not always, with the result that we are dealing with an active

bacteremia and many times this is quite impossible to control. A sterile blood culture with a constant temperature, regardless of the cause of this temperature, usually means a fair prognosis. Like the sterile blood culture, the negative spinal fluid, when cerebral symptoms are making their appearance, is always reassuring. However, the presence of an increased number of polynuclear leukocytes in the blood is forewarning of an infected meninges and no time should be lost in finding and eradicating the focus of infection.

DR. MAX A. GOLDZIEHER: It was exceedingly gratifying for a laboratory man to listen to Dr. Kopetzky's paper, which stated how much help can be derived from the aid of the laboratory for clinical medicine. I was even more impressed by the statement that the laboratory should be considered only as an aid and that the clinician must use his judgment and experience to decide the case. It would be a mistake to consider laboratory tests as final. I think you will all agree with me on that point, and I will give you a few instances of how laboratory tests, although made very accurately, might be misleading. Let us take, for instance, a red cell count. If you do it repeatedly on any normal person you will find a vacillation of the red cell count, not of 10,000 to 20,000, but differences in the counting which run into 100,000 in the same normal, healthy person. It is the same with the white cells, and if you want to get a better view of the matter, remember the work carried out by Peterson in Chicago, and Mueller in Germany, which has shown that very slight irritations of the skin are liable to produce very considerable changes in the blood. We have to remember that the actual quantity of blood cells in the circulating blood depends on two factors; first, their liberation from the bone marrow; and second, their distribution in the different parts of the vascular system, such as the abdominal vessels under splachnic innervation, or the peripheral blood vessels. This distribution is subject to a great many different influences.

Another point: Hemolytic streptococci, or staphylococci, if located somewhere in the tissues attract the polynuclear leukocytes. Yet if you change the diet of your experimental animals, inasmuch you put them on a high proteinic food and give them but very little water, you will not find any leukocytes aggregating around the bacteria. Necrosis of the tissue is the only reaction, or you may get a proliferation of lymphocytes and macrophages.

The same thing can be observed in the human in case of the so-called agranulocytic angina. The polynuclear leukocytes disappear from the blood in these cases; and in the foci of infection, such as tonsils or cervical lymph nodes, you will find necrosis surrounded by lymphocytes and macrophages, but not a single polynuclear. In one word, an identical picture as that of the animal experiments quoted.

These points were brought up to show that the presence of a certain infectious agent in the human body will not be followed always by the same biological reaction, as such reaction depends on a great variety of factors, only a part of which is known so far.

If you will bear all this in mind you will see how many sources of error are liable to enter into the laboratory findings, but if they are used with care and judgment by such a master as Dr. Kopetzky they are very helpful; not if they are used superficially and with a mind that is too lazy to find out whether they are substantiating the clinical findings obtained at the bedside.

DR. H. K. TAYLOR: I will refer to only one part of this paper, the radiographic end. There are some things which are essential in the interpretation of the Roentgenographic plate, and I most heartily agree with Dr. Kopetzky that an X-ray plate is only an aid and not a diagnosis. I have had a great deal of difficulty in impressing that fact. The anatomy as demonstrated on the Roentgenogram is of considerable importance to the otologist—and the features demonstrated there, *vis.*, the size, the extent of the cellularization ,the extension of cells into the squamosa, the occipital bone, the petrosa and squamosa; the character of the structures—whether small or large cells, or sclerotic; then the situation of the sinus plate, where it is located, the condition of the sinus plate, the condition of the tegmen; and the anomalous mastoid which we meet at times . The mastoid processes in most individuals are alike, but there are cases where they vary in size and shape and are very misleading. Then, again, the presence of a mastoid emissary vein is to be noted.

I will not touch on the pathology for Dr. Kopetzky has covered that exceptionally well. In cases where the destructive changes are very small, in a large mastoid, it may take a long time before you get definite clinical symptoms which would necessitate operation. I have seen many cases that were almost asymptomatic. In a small mastoid these same changes will give considerable clinical symptoms.

The situation of the sinus plate; we have observed that the forward-lying sinus is quite superficial and should be known to avoid injury. The importance of the mastoid emissary vein is well known. Another point that Dr. Kopetzky brought out is that in the mastoid when you find a destructive lesion it is very important to determine whether the ear has been the site of a previous otitic infection. In many cases, a destructive lesion may be due, not to the present, but to a previous otitic infection—a diagnostic point which is not very well known. Another is that the sclerotic mastoid may sometimes be confused with a coalescent mastoid. In a sclerotic mastoid, the bony portion of the Eustachien tube is usually visualized.

I may finish as Dr. Kopetzky did—that the clinical findings and not the Roentgenogram should be the chief guide to the clinical procedure.

DR. F. M. LAW: I think Dr. Kopetzky's paper is the most concise and complete resume of the Roentgenographic findings in the mastoid that I ever heard. Every word said is absolutely true, and I would like to emphasize just one point—that is, that the surgeon should make his own interpretation, with the aid of the Roentgenologist who made the plates. The mere clouding of the mastoid area means nothing; the true meaning lies in the character of the mastoid cell structure itself—whether the cell walls are edematous, whether thick or thin from pressure, whether there is or is not absorption or softening of the cell walls. The clinical knowledge, coupled with the picture shown (provided it is a good one) is the only way in which you can get real knowledge from a Roentgenogram. A technically poor film is worse than useless. I consider a technically good film to be one which shows clearly the cancellous structure of the bone surrounding the mastoid.

DR. KOPETZKY: I may only add that I have limited the paper on laboratory aids to those with which I have had experience; the additional aid which Dr. Ottenberg brought out is one with which I have had no experience at all. I only expressed my experience, and I wish to thank you all for your kind indulgence in listening to it.

The Effect of Sinusitis on Certain Syndromes of Chiasmal Tumor. Dr. H. I. Lillie and Dr. Walter I. Lillie.

(To appear in a subsequent issue of THE LARYNGOSCOPE.*)*

DISCUSSION.

DR. WELLS P. EAGLETON: When the Chairman asked me to discuss this paper I told him I knew nothing about the subject, but Dr. Lillie kindly sent me his paper and after reading it I was still more convinced that I knew nothing about it—but this paper is of the utmost importance to us all. Dr. Lillie has brought forward cases in which there is a combination of sphenoid disease with chiasmal tumors. Now, Dr. Cushing objected to every man who was going blind going to an ophthalmologist and being asked if his nose had been examined on being told no, being sent to a rhinologist and having his sphenoid opened; and with a full grown tumor which Dr. Cushing claimed should have been diagnosed at the time of the nasal operation.

There have come under my observation seven cases in which the sphenoid has been opened for approaching blindness. Three of them were immediately relieved because they were properly diagnosed—the effect was marvelous. The other four were as follows: 1. Injury; after carefully questioning the man there was no doubt that he had alcohol emblyopia; of course opening the sphenoid would not help that. 2. A typical case of vascular occlusion; the man suddenly went blind in one eye; nothing seen. Going to an ophthalmologist, he was sent to a nose man, who opened the sphenoid. Examination of the heart showed that he had an organic lesion, and there is no doubt that he had vascular occlusion. 3. A man with an intracerebral condition, as shown by my coming

later on to do a temporosphenoid operation. I tapped his ventricle and found an enormously distended ventricle. 4. A typical case of aneurism of the intercommunicating artery which had been, more or less, an intranasal operation.

Now, out of these seven cases, three of them justified operation; and in four of them the surgeon threw discredit not only on his own diagnostic ability but also on surgery.

I cannot add anything to what has been said. The demonstration of the fields was a wonderful demonstration of what should occur in every rhinologist's room. Not only should a case be examined rhinologically, but ophthalmologically, especially in relation to the fields; and then a thorough neurological examination should be made before an operation for the relief of blindness.

I may add a word or two on the diagnosis of the different conditions. I think the whole proposition of diagnosis rests on one little trick—that is, that the optic nerve goes through a hole that holds it firmly—the optic canal, in fact. It depends on the lesion, whether it invades that place or not, whether we are going to have any relief from intranasal operation, whether it has anything to do with the intranasal sinus. In many cases going blind, it should be remembered that the optic nerve going through the canal is adherent to the canal itself. If you examine the head of a cadaver and remove the brain, with the nerves lying all before you, and lift them, you cannot pull the optic nerve back into the brain; they are firmly adherent to the wall of the optic foramen. Now, if we have cellular disease, the cellular tissue around the optic canal is inflamed, and as the dura is adherent to the bone, it is also inflamed, and you have a round cell inflammation immediately; it cannot be otherwise. Now, if you open the sphenoid in such a case you will immediately give relief, provided the sphenoid is opened promptly. Because why? Blindness from sphenoidal diseases does not wait long, because it is a blindness from the nerve, and blindness from chiasmal tumor is due to pressure; and in all cases that I have seen, pressure on the eye ceased after operation in true sphenoidal disease. Pressing on the eye, you are pressing backward toward an inflamed surface in the brain; and, another thing, the movement of the eye itself is painful because the nerve is adherent exactly in the place inflamed. That is of the greatest importance to me. If I press the eye and it does not hurt them, it is not sphenoidal disease, but if it does hurt, I think it may be spheioidal sinus disease.

DR. LOUIS HAUSMAN: I am very glad to have had this opportunity of listening to the interesting material which Dr. Lillie has so beautifully presented. The problem is one of great importance and concern to the neurologist, as well as to the otologist and rhinologist. Perhaps I can best contribute to the discussion by elaborating upon several pertinent points of special interest to the neurologist.

The question of the acuteness of the amaurosis is frequently a very perplexing one. It may be acute or not, according to the interpretation of the history. What I mean is best illustrated by a case, I recently observed, of a woman with various congenital defects, who was admitted to the Neurologic Service at the Mt. Sinai Hospital with a history of acute blindness in the right eye. A careful study of the visual fields revealed complete amaurosis in the right eye, with a temporal field defect in the left eye.

In other words, the suspicion arose that the blindness in the right eye really was not acute, but that the patient had failed to notice an antecedent hemianopic defect in that eye. In fact, an opthalmologist at an earlier examination had made the diagnosis of a retrobulbar neuritis in that eye. In many of these cases the patient may complain of acute blindness in one eye, whereas the condition actually has been of long standing, but the patient has failed to recognize the existence of an earlier partial field defect in that eye. As soon as the rest of the field is impaired, the patient's impression, of course, is that of acute blindness, which is misleading, for the underlying condition may be a neoplasm of the optic chiasm or nerves. It is not at all uncommon to find in routine examinations a bitemporal hemianopsia or homonymous hemianopsia, of which the patient was entirely unaware. For that reason, a history of acute blindness must be accepted with great caution.

Another difficult problem concerns the cases of retrobulbar neuritis, in which the suspected sinuses are opened and although no pathologic process is found,

the retrobulbar neuritis disappears. What condition are we dealing within this type? The possibility of multiple or disseminated sclerosis must be borne in mind, for retrobulbar neuritis may be the first symptom of the disease, only to disappear and be followed later by the other signs which make the diagnosis of multiple sclerosis more definite. Many cases could be cited bearing upon this point. They are not of a toxic nature, for the lesion is not bilateral.

It has been suggested that the bleeding attending the opening of the sinuses is the helpful factor, but I doubt it. It is curious that purulent sinusitis so seldom involves the optic nerve. I should like very much to hear from Dr. Lillie of his impressions along these lines.

DR. JOHN M. WHEELER: The hour is late and I shall not drag out the discussion but shall simply make two points. One is closely akin to the point Dr. Eagleton just made. The rhinological surgeon should not be ignorant in regard to the fields of vision. I think the nose surgeon should have a right to ask the ophthalmologist what his findings are in such cases as Dr. Lillie has discussed—to ask whether the fields have temporal defects, whether central scotoma, or other significant findings, and should ask the eye man to explain what is meant by these findings.

That leads me to the point that I think the nose surgeon should not be a surgical mechanic for the ophthalmologist or for the neurologist; he should be the supreme judge himself as to whether operation should be performed, and how it should be done, if he deems an operation wise.

Another point is that there is an important condition which has not been spoken of tonight, which is rare but very important; that is mucocele of the sphenoid sinus. I have seen one case. In 1921, Van der Hoeve, of Leiden, Holland, reported before the Ophthalmological Society of the Netherlands a case of mucocele of the sinus and described it in detail. It was diagnosed as tumor, probably in the region of the chiasm. Van der Hoeve himself operated on the case through an incision in the orbit and explored with his finger, to see what he could find. He described his findings, which I will not attempt to give in detail, but there was a bulging of the rear portion of the nasal wall of the orbit; this led to an operation on the sphenoid and the mucocele was found. This condition is one in which the communication between the sphenoid sinus and the nose does not exist—either never has existed or has become occluded, and the contents of the dilated sphenoid cavity consists of mucus, some degenerated cells, some white and some red blood cells, but principally a quantity of choleterin crystals.

Dr. Van der Hoevre pointed out that eight cases of mucocele had been described in the literature—nine casees, including his own—and in not one instance had the proper diagnosis been made before operation. In view of that, although the condition was considered rare, it is probably not so rare as it is considered; and otologists and rhinologists should bear in mind the existence of such a condition, which may produce signs and symptoms akin to those described tonight. In one case which I saw a number of years ago at the Cornell Clinic optic atrophy was manifest; one eye was blind and in the other the sight was impaired. We did not make the diagnosis, but an exploratory examination of the nose was made by Dr. S. L. Craig, and in attempting to probe he broke through the wall of the sphenoid and out gushed a quantity of chocolate-brown fluid, and in the laboratory a quantity of cholesterin crystals were found. I never described the case, for I allowed neurologists to take the patient, and I never got it back.

Let me say once more that the condition is sufficiently important to be borne in mind, considering the type of cases discussed tonight.

DR. LILLIE (closing discussion): The subjective pain on motion or resulting from manual pressure on the eyeball in cases of optic neuritis, mentioned by Dr. Eagleton, is very interesting. I have had no experience with this type, but a few of our proven cases of meningitis associated with choked discs have had a subjective photophobia when examined ophthalmoscopically.

Dr. Hausman's experience coincides with ours, in as much as the greatest number of unilateral central scotomas associated with a normal fundus, have proved to be cases of multiple sclerosis, especially between the ages of 12 to

40 years. Recently in the French literature a case of unilateral central scotoma in a girl, age 26 years, was reported cured by deep Roentgen ray therapy. It makes one wonder whether the condition was a prodromal of multiple sclerosis. In cases of frank sinusitis ophthalmologic complications are extremely rare in our experience.

The case mentioned by Dr. Wheeler of a mucocele of the sphenoid sinus producing a central scotoma just shows the great diversification of pathology that can produce this picture. A careful history, stressing the type of onset, progression, regression, et cetera, are of great value, combined with the objective findings, in correctly designating the basic pathology. Personally, I have had no experience with a case with mucocele of the sphenoid sinus.

Again, I wish to thank you for the privilege of being your guest, and regret that my brother could not be here to enjoy your hospitality.

SECTION ON OTOLOGY.

May 11, 1928.

Unusual Case of Chronic Mastoiditis. Dr. John Horn.

A case of bilateral otitis media purulenta chronica, chronic mastoiditis. Right ear with a fistula in the outer border of the posterior wall of the external auditory canal. Simple mastoid operation. Recovery.

The patient was a girl, age 14 years, who gave a history of having had discharging ears for seven years. Examination, Oct. 16, 1926, showed both canals filled with a foul-smelling secretion, drum membrane lacking, no ossicles; the outer border of the posterior wall of the right external auditory canal showed a fistula containing thin pus. This was carefully probed, the probe entering a cavity. A crucial incision was made through the opening and with a blunt curette some diseased bone was removed; a simple mastoid operation was done three days later. The bone was found to be markedly sclerosed and deep layers of cells diseased; thorough curetting, especially in the neighborhood of the fistula. A small area of the sinus was exposed during the operation. The wound was drained with gauze strips and closed with catgut sutures. Uneventful recovery. The patient was discharged from the hospital after three days.

The post-operative treatment of the otitis media purulenta chronic of both ears consisted of applying unguentum Crede (Horn). The right ear was dry after six weeks; except for the fistulous opening there were no other symptoms. The left ear occasionally shows a slight, colorless discharge. The hearing in both ears has greatly improved.

DISCUSSION.

Dr. Clarence H. Smith: Dr. Horn has shown us an interesting case with a very good result. I would like to say something about my experience with Dr. Horn's method of treatment with unguentum Crede. I have been using it in chronic purulent otitis media for some months and outside of operative measures find that it is the best therapeutic aid I know of. I tried a series of 20 cases with zinc ionization, but did not get one cure. I have tried all kinds of different dyes and powders, a powder with iodin in it, boric acid powder, etc. I had some good results with the idoin powder, but it proved to be rather painful. The unguentum Crede I have used with very startling results. I don't know how it works. Dr. Horn says it is a powerful bactericide, that it is based on collargol and to that owes its virtue. I have had some cases where the ear had been discharging for 20 years, that dried up after two or three treatments, and anything that will do that is well worth having in your armamentarium. It is not painful, and outside of being rather messy in appearance —which does not amount to anything—I know of no objection to it. I do think that perhaps any long method of treatment carried out by the doctor himself, as this is, every second day, might bring results in some cases, but that does

not explain some of these old cases that dry up after two or three treatments, cases which previously had been in good hands and under all kinds of treatment. Dr. Horn has discovered something very useful in this method.

DR. HELLER: I have had no experience with this treatment and am not competent to criticize or discuss it. I did not intend to say anything until I heard Dr. Smith say that he got no results from ionization. I am very well pleased with that method and have had some good results from it,—in one case, that had been under my observation for 14 years and was also seen by Dr. McKernon, who advised a radical mastoid operation, which I also urged, but the patient refused. She was a young woman, of about 30 years, who had been treated with all the ordinary routine measures, two or three times a week under close observation, without healing; but when ionization came out two years ago it was tried and the ear has been dry ever since.

There is nothing more tricky that we have to deal with than chronic otitis; there are many factors that have to be taken into consideration in chronic running ears—pharyngeal affections and infections of the nose and throat. Dr. Horn has shown this case tonight, but I would be willing to bet that in the next two or three cases he would not get the same good result. In this particular instance Nature has done a good work in establishing a fistula and good drainage, which, after all, is the crux of surgical procedures where there is pus. I think that most of these chronic ears are due to the fact that the drainage is poor. Dr. Gottlieb will remember a case we saw some 15 years ago—a young woman with the entire inner process reamed out by Nature and she got well as if by a radical operation. The method which Dr. Horn has described is at least rational; it is true antiseptic treatment and I shall be glad to try it.

DR. HORN: A physician brought to me a relative, age about 70 years, who years before had undergone a radical mastoid operation on the left ear at a European clinic, and who had a secondary radical mastoid operation two years thereafter, but which was still unhealed. When I examined the patient I found her suffering from a foul-smelling discharge from both ears, and there seemed slight hope for any improvement. She is well today.

Now, as to drainage: There is no obstruction to drainage; the middle ear cavity is open; there is no way of stopping the exit of pus. The foulest-smelling ones have surprised me more than those with small open fistulas. I have seen a large perforation and a polyp—removed the polyp with a snare, treated the stump with nitrate of silver, 10 to 20 per cent, and had a splendid result; and the hearing was very satisfactory, to my surprise. I would suggest that this treatment be used also in slow-healing postoperative chronic mastoid cases.

Short Reports of Five Interesting Cases of Chronic Mastoiditis. DR. Clarence H. Smith.

DISCUSSION.

DR. MARK GOTTLIEB: We ought to congratulate Dr. Smith on the presentation of such interesting cases; every one is worthy material for discussion. I think that during last fall and winter we have seen more severe cases running through more severe crises than for several years previously. The last case shown by Dr. Smith is very unusual, and the child's recovery was largely due to his good judgment, for which he is to be congratulated. Last year I had a case of a child with tonsillitis, who then developed an acute otitis from which she was recovering, when suddenly one day the temperature rose to 104°. There was a scant discharge from the ear and no outward signs of mastoiditis. A pediatrist was asked to see the child, but could find nothing. The mastoid, therefore, was under suspicion. We were not satisfied with that decision and called in another pediatrist, and finally decided that the ear should be opened on suspicion only. When opened, the mastoid was found to be completely destroyed and the sinus plate was eroded. The temperature was 103.5° at the time of operation, but the next morning it was normal, and at 11 o'clock of that day the temperature was 106° and the pulse could not be counted. The child was age 3½ years. We were very much alarmed about it and felt that we were dealing with a sinus thrombosis and that it was necessary to do some-

thing. We had several other consultations and decided that evening to give a blood transfusion, ligate the jugular and obliterate the sinus. We did not find a clot, and the temperature came down gradually, it being septic for a week-and-a-half. After this the child was sent home. Then the mother developed tonsillitis, and transmitted it to the child. While recovering from this she developed as large a retropharyngeal abscess as I have even seen. This was incised and the child proceeded to get well without further mishap.

DR. HELLER: I think Dr. Smith's judgment in giving the child transfusions was absolutely right and excellent. It is a very valuable adjunct in our septic ear cases. We are doing it more and more, and a number of lives have been saved by it. In many cases where you feel by the appearance of the child and everything that there is some absorption from the wound, I know of nothing better than a transfusion, repeated two or three times if indicated. I think that two of Dr. Smith's cases were saved by the transfusions.

DR. S. M. JACOBS: I join the other speakers and congratulate Dr. Smith on his able presentation of these very interesting and highly instructive cases. There is no doubt that blood transfusion is a great adjuvant in these septic cases. I wish to cite a case which is under my care in Lebanon Hospital at present. A girl, age 9 years, has been suffering from bilateral otitis media purulenta acuta, three weeks following measles. The temperature ranged from 99° to 103°. The child looked and acted well. The classical pathognomonic symptoms of acute mastoiditis were lacking. There was no tenderness or pain on deep pressure over the mastoid, nor was there sagging. But there was something in the appearance of the middle ear which made me suspicious of mastoiditis. I performed a double mastoidectomy and found extensive necrosis, more so on the left side. Temperature rose to 105° and remained stationary for two days. Blood culture positive, two colonies to 1 c.c. Another culture returned positive, four colonies to 1 c.c. I advised ligation of the internal jugular and to explore the sinus. Two well known otologists were called in consultation and, to my surprise, both, in spite of the positive blood culture, advised waiting. The temperature ran a septic course, 99-106°. Hemoglobin went down to 50. Two blood transfusions of 250 c.c. were given. Four subsequent blood cultures were sterile. On the seventh day I decided to ligate the internal jugular. Here I was confronted with a difficult problem—which sinus to explore and which jugular to ligate. In view of the fact that the left mastoid presented more pathology, I decided to enter the left side. But before doing so, I exposed both the right and left sinus to see which presented more pathological changes. Neither showed any. I tied the left internal jugular and opened the left sinus and found a septic clot at the bulbar end. The child's temperature came down, but went up again to 104°; hemoglobin, 45 per cent; and another blood transfusion of 225 c.c. was given. Temperature subsided and for the past few days remained stationery at 99°. I cannot emphasize too strongly the value of blood transfusions in these septic cases, not so much for the bactericidal properties, but for its supportive action to maintain the proper amount of hemoglobin and red blood cells, thereby increasing the resistance of an already debilitated patient.

The Finding of Contraction of the Visual Field in Progressive Deafness, with Report of Cases. DR. MARK J. GOTTLIEB.

DISCUSSION.

DR. AGATSTON: I have had the good fortune of examining the cases which Dr. Gottlieb is reporting. The result is suggestive of a relationship between the degenerative process of the ear apparatus and degeneration of the optic nerve as evidenced by the contracting of the visual fields in his cases. Of course, we have not had a sufficient number from which to draw definite conclusions, but certain it is, that visual fields must be taken in these cases and at some future date we may be able to make a more positive statement. Our findings correspond to a partial atrophy of the optic nerve, involving the central fibres of the nerve, though the papillomacular bundle seems unaffected. The reason that we localize the trouble in the centre of the nerve is, because in a general contraction of the peripheral visual field, it is the centre of the nerve

that must be affected, since the central fibres are distributed radially to the periphery of the retina, while the peripheral fibres represent the area surrounding the disc, as illustrated in the schematic drawing. In the event that a distinct correlation is established, since this type of optic atrophy is generally caused by some form of toxemia, one would infer from the eye findings that this disease of the cochlea is also caused by some form of toxemia. In none of our cases have I found atrophy sufficient to produce abnormal pallor of the disc. The appearance of the fundi was uniformly negative.

In taking the fields, I was very conservative and tried to make them as large as possible. I used a 1 c.m. test object, and when the field was contracted, I also used a 2 c.m. object, to relieve any doubt in my mind as to whether the patient was co-operating. Diagram of the optic nerve was demonstrated on the screen.

DR. JOHN GUTTMAN: We must appreciate highly the great amount of painstaking work required in the examination of these cases. While I do not consider myself competent to judge the diagnostic value of the blood chemistry in these cases, still I believe that the clinical symptoms described could very well fit in a picture of adhesive otitis media or conduction deafness caused by nose and throat affections, with the exception, perhaps, of one case where a gastrointestinal autointoxication could have caused a perception deafness.

The lantern slide demonstration of the limited field of vision in these cases was not marked, and was not very convincing, either. The introduction of a new nomenclature, as constitutional deafness, for a clinical picture which is complicated enough by numerous designations, I do not consider very fortunate.

DR. GOTTLIEB: I have read many times of an individual who was having his hearing examined and the diagnosis was said to be one thing and it turned out to be something else. I cannot establish a diagnosis of otosclerosis. I believe the autopsy would be able to tell that, but I am not hoping that my patients die so that I may examine the petrous portion of the temporal bone. If you examine a large number of patients consecutively you will find a great variation in hearing; the value one day will be entirely different from that on another day. The finding of the value of the contraction of the visual field so regularly as we have found it, and of others who have found a similar proportion, would be unusual unless there was some relationship. If a toxemia can produce a neuritis of the central fibres of the optic nerve, manifested by contraction of the fields of vision, it certainly can produce a lesion in the cochlear nerve. This is even more probable when we consider that the circulation in the modiolus is such as to lend itself to such a lesion.

I have presented the result of certain examinations, and deductions have to be made if we are going to advance. At the same time, certain facts would seem to justify these ideas.

DR. AGATSTON: It seems to me, when considering the optic nerve in correlation with the acoustic nerve that it is possible to have an analogy. There is no reason why the acoustic nerve cannot have a degeneration of central fibres, as well as the optic nerve. As I mentioned before, we have not as yet had a sufficient number of cases from which to draw definite conclusions, but we feel that we have enough to warn the otologist that in cases of so-called otosclerosis with open Eustachian tubes, visual fields should be taken. As to the accuracy of the fields—I was careful to take them so many times (usually in the morning) that if contraction was shown it was real. My normal limits were low: Temporal, 70°; nasal, 50°; inferior, 50°; superior, 45°. Normal fields for red: Temporal, 40°; nasal, 30°; inferior, 25°; superior, 25°.

SECTION ON LARYNGOLOGY AND RHINOLOGY.

Meeting of May 23, 1928.

Nasal Polypi in an Eight-Year-Old Child; Case Presentation. Dr. Alfred Michaelis.

Tamponing the Nose. The Use of Tampons in Ordinary Rhinological Work. Dr. John Horn.

As applications to mucous membranes of the nose are rapidly washed away by the natural secretions and where a continuous action or prolonged effect is desired, it occurred to me to try tamponing the nose by a series of four cotton pledgets about 1 inch long, lightly folded and tied to a strong cotton thread sufficient in size to be readily inserted into the nose, each separated from the other about 3 inches, the appearance of these tampons being similar to the old-fashioned "kitetail".

The first of the pledgets is to be saturated with one of the many medicaments in use for the appropriate pathological conditions present, then inserted into the upper nasal fossa, followed by the succeeding three tampons, filling the entire nasal cavity and kept within the nasal orifice in such a way that it is not noticeable from the outside. Applications thus made under direct inspection will reach large portions of the nasal mucosa.

The tampons may readily be removed by the patient after a certain length of time, as advised by the attending physician.

Burnt sponge-tinder, Boletus Igliarius Feuer schwamm or zunder derived from an excrescence on oak or beech trees in Germany and used as a hemostatic.

Since writing this article I have been informed that a method similar to this has been employed by Dr. Joseph C. Beck, of Chicago. I am not acquainted with his exact technique.

DISCUSSION.

DR. HARRIS: I think Dr. Horn should be complimented on the ingenuity he has displayed in this method of application. The use of sponges for the purposes of controlling bleeding I have had no experience with. There were other methods which would seem to me fully as good, or better possibly, but I think there is no one of us here who has not had the embarrassing experience of putting cotton in a nose and having the patient tell us that he got a piece of cotton out of his nose a week after, or had a bad odor for a time, so that this method we can welcome and make use of.

Normal and Pathological Anatomy of the Maxillary Sinus. Dr. Geo. W. Mackenzie.

DISCUSSION.

DR. G. W. MACKENZIE (closing the discussion): I want to thank the gentlemen who discussed the paper, and assure them that it is appreciated. I want to apologize to those gentlemen whose names appear on the program for discussions of the paper for not having sent them a copy prior to the meeting. The reason for it is that the paper was not completed until today. Besides, the subject is so fundamental that I thought it was hardly necessary.

As to the use of lipiodol in the diagnosis of dentigerous cysts, referred to by Dr. McPherson, I am passing around an illustration of a case where lipiodol was used for the purpose suggested.

Regarding the diagnosis of bacteriological washings from the maxillary sinus in order to determine from the recovered secretions what bacteria are present, I prefer not expressing an opinion at the present time. It is too big a subject to discuss in a few minutes. There are so many technical points about it. For instance, when washing out the maxillary sinus it is difficult not to include some of the bacterial flora present in the nasal cavity. Bacteria are almost always present in the nasal cavity, surely in all nasal cavities that are infected. I believe it is quite possible for an X-ray man of the capability of Dr. Law to determine quite accurately the outline of a cyst by the slight, but definitely increased density of the epidermal layer of the cyst which was easily recognized in all the cases I have seen.

I recall the first Roentgenologist with whom I collaborated in Philadelphia, failed to recognize the cyst until I pointed it out to him. After that he never failed to recognize a dentigerous cyst. I am very strongly in favor of Roentgenological examinations, particularly by the one who knows how. I find the services of Dr. J. W. Post, of Philadelphia, indispensable to me. I hesitate to come to a positive diagnosis in any ayptical maxillary sinus condition without his assistance. He submits an unbiased report and I find that he is generally able to make a diagnosis that checks up with my clinical diagnosis and operative findings.

As to what is a normal maxillary sinps: I think the doctor who raised the question answered it himself. As to going into the ethmoids by way of the maxillary sinus route, there are some cases in which it is impossible. For instance, in those cases of excessive outward bulging of the lateral nasal wall, particularly of the middle fossa, where one comes directly onto the orbit. As regards other cases, if one knows in advance the anatomical relationship he can select the cases where this particular procedure can be applied.

The older writers thought that the majority of cases of maxillary sinus disease were secondary to infections about the teeth, and since Zuckerkandl's work the number of such cases has steadily dwindled. There are many reasons why one should suspect the teeth as the etiological factor in mavillary sinus disease, which were referred to in the paper. It is remarkable how many periapical abscesses we find present by X-ray examination and by cultural methods which after removal show no evidence of sinus disease, in spite of the fact that many of them reach well up into the sinus through its floor. However, where the dentist, in an effort to be clean, syringes out the socket, resulting in flushing infectious material into the maxillary sinus, in most cases infection of the sinus follows just as surely as douching the ear in the case of traumatic rupture of the drumhead results in middle ear suppuration.

DR. DUNCAN MCPHERSON: I am very glad that I have had the opportunity of listening to and taking part in Dr. Mackenzie's paper. However, I came intending to discuss diagnosis. Unfortunately that was not touched on in the paper. I felt that the pathology and the anatomy would be taken care of by Dr. Eggston and some of the pathologists present. I was a little bit frantic when I found there was nothing to be said on diagnosis, so I tried to make some notes, and now I cannot read the notes! Being inclined towards practicality, I did try to make notes in an endeavor to apply some of the things which Dr. Mackenzie brought out in his pathological demonstration. In children, especially in young children, on account of the thickness of the bone down towards the floor of the nose, the antra may be difficult to enter. Dr. Mackenzie referred to dentigerous cysts, and these of course you all know we may not see in radiographs. He did not say anything about the use of lipiodol, but he was not talking about treatment. Occasionally lipiodol will help to outline the septa that he showed developed in the nose, and perhaps outline some of the smaller as well as larger osteomata. I would like to ask Dr. Mackenzie, although he has not touched upon it tonight, whether bacteria are not usually found in the normal antra and what value would be obtained in making cultures from centrifuging in antral cases, granting that the sinus is likely to be involved in a bacteriological way very much much as the nasal mucous membrane would be involved, to some extent at least.

A friend had a radiograph made of a patient, in which there was a large opening in the sphenoid, and the radiographer claimed that he could make a diagnosis of polypi in the sinus. My friend took a polyp from the ethmoid and put it in his sphenoidal sinus and then sent the patient back to the X-ray man for examination. It came back normal sinus, the same as originally, so that in a practical way polypi are not always discoverable by X-ray. I feel that I am straying a little from the anatomical and pathological phase of the subject, and yet in a practical way I might cite another case in which a picture that the doctor showed demonstrated an apparent horizontal septum of the maxillary sinus, in which, however, the upper chamber was an ethmoid and drained posteriorly, and the lower chamber was the true antrum. In a case that I had I made two openings and I made a diagnosis of a double antrum with a low roof and a floor to the upper chamber, going in above and below, failing to get

anything below, and then went in above and got pus coming out from the upper chamber. Whether it came out in front or behind I am unable to say.

DR. FREDERICK M. LAW: I have thoroughly enjoyed this paper of Dr. Mackenzie, because from the Roentgenologist's point of view it has been exactly the demonstration I have been favoring for a good many years. Personally, I feel that the value of the X-ray of the sinuses is far more useful as a demonstration of anatomy than it is of pathology. The things which Dr. Mackenzie showed tonight are perfectly demonstrable with a properly made Roentgenogram. When I say "properly made" I means that the technique should be perfect, not a poor plate, but one which shows detail sufficiently to be able to determine the cancellous structure of the bone. So many times we see films presented for interpretation in which one can scarcely see the gross anatomy, and I have seen interpretations given from such films.

The recesses in the alveolus are much more frequent, I think, than we realize. In thousands of examinations which I have seen at the Manhattan Eye and Ear Hospital, recesses even to the extent of the largest which Dr. Mackenzie has shown tonight are quite frequent.

Dr. McPherson spoke of polypi. I remember that case very well. There is no excuse for it at all, but it was a good many years ago, and I do not think we would see that same polyp today. If he had taken the polyp and placed it in the sphenoid from which a polyp had been removed and in which the surrounding mucous membrane and structures were still edematous, we might have been able to make a different interpretation than a normal sinus, but this does demonstrate the fact that pathology is not always grossly shown on a sinus plate, and the information which gives the most good is that derived from a plate which not only shows the gross anatomy, but the minute structure of the mucoperiosteum, as shown by the microscopic slides tonight, and in a great many cases an interpretation can be made from a properly made sinus examination which under the microscope will show the same changes that Dr. Mackenzie showed on the slides, and I think this demonstration tonight adds just another link to the chain of evidence which I have tried to get laryngologists to realize: the importance of conferences with the Roentgenologist in order to make good interpretations. The laryngologist, with his knowledge of the pathology and symptomatology and the evidence derived from his talk with the Roentgenologist is able to get information which the Roentgenologist alone cannot do, and so many times the Roentgenologist has to make an interpretation from a plate on which he has no information of the symptomatology or clinical evidence, and we cannot make any interpretation without complete clinical evidence.

I want to thank Dr. Mackenzie for bringing up the subject of anatomy, which, from the surgeon's point of view, is far better than pathology.

DR. MCPHERSON: I am sorry that Dr. Law remembered that case, but I would like to emphasize the importance of not accepting the very poor plates that are coming to us from so many sources. It is our duty as laryngologists not to accept those plates. We are getting them from all quarters, and it is absolutely impossible to get from them the information we want. I have had them from commercial houses and from some members of the profession. So long as we accept poor plates, so long will they be made.

DR. LEON T. LEWALD: I agree with what Dr. McPherson has just said and what Dr. Law has said about obtaining sufficient detail. Personally, in some cases I prefer very much to have a dense exposure made with a Bucky diaphragm to bring out such a condition as a septum in the antrum. I also use stereoscopic views and find that they are a decided help in doubtful cases. Occasionally, also, I have been able to diagnose a dentigerous cyst, and even in one case a polyp, the diagnosis of which was concurred in by Dr. Huey at the New York University Clinic, and the dentigerous cyst was confirmed by operation by Dr. Huey.

DR. MICHAELIS: The point which interested me greatly was the differentiation between the region of origin of simple and cystic polypi, namely, that the simple polyp is apt to spring from an area removed from the vicinity of the sinus ducts or openings and that the cystic polyp always grows near the orifice. This statement throws some light on the case I described earlier in the evening,

and where I made the clinical diagnosis of ethmoiditis of the posterior cells because the polypi appeared to spring from the natural drainage area of these cells. This differentiation is a fact I did not know.

DR. LEOPOLD GLUSHAK: From what we hear tonight, it seems to me that we still have dodged the question, What is a normal antrum? and, unfortunately, all the various anomalies have hitherto not been classified. From the operative point of view we discriminate when we enter the antrum through the anterior wall. We have to be aware of the existence of various septae and recesses, and it is difficult unless one exposes very thoroughly, to know where the antrum ends, in spite of the fact that it has definite walls. There is one point which I think might be brought to your attention from a practical surgicoanatomical point of view, and that is the position of the floor of the ethmoid in relation to the antrum. Operating through the antrum, where we have a definite involvement of both sinuses, and even going far back to the sphenoid, particularly in cases of ozena, where you attempt the Lautenschlager procedure of displacing the lateral wall toward the septum, thus narrowing the nose, the floor of the ethmoid is observed to be rather hidden in an oblique position above the natural opening of the antrum.

As we enter through the canine fossa, we find a bowing in of the inferior meatus into the interior of the antral cavity, high up is the natural opening, above which we have the somewhat oblique roof as it merges into the floor of the orbit. Unless one looks very carefully for this slanting part of the roof, one cannot find the floor of the ethmoid. This is usually found if you look around the corner in an upward and medial direction over the inferior meatal bulge into the antrum. The latter is an anatomical variation; in some it shows an exaggerated convexity into the antrum, so much so that one is apt to go into the inferior meatus during the initial chiseling through the canine fossa, mistaking it for the antrum, which is laterally displaced. The danger of getting into the orbit is obviated by the X-ray plate. We must have an X-ray picture beforehand to be sure that the ethmoid does not come to a narrow base; as one can easily get into the orbit instead of getting into the ethmoid. In the majority of cases one enters the ethmoid very easily, and can clear out all the cells with the exception of the very anterior ones, which can only be reached intranasally.

SECTION OF OTOLOGY.

Meeting of Friday Evening, October 19, 1928.

Chronic Mastoiditis and Labyrinthitis Following Gunshot Wound of the Ear; Operation; Recovery. Dr. John McCoy.

Harry P., Greek, age 32 years, was admitted on May 13, 1927, to my service at the New York Polyclinic Hospital.

History: After backing his auto into his private garage one night about two months previously, he was held up by gunmen for money and valuables which he had in his possession. After being told to throw up his hands he was shot in the right ear. He described his first feeling as one of tremendous dizziness, and this was followed by unconsciousness. He was brought to the Nassau General Hospital and remained there from March 14 to May 6, during most of which time he was in a very serious condition, at first having exudate of cerebrospinal fluid.

On May 13, 1927, he entered the Polyclinic Hospital with the preceding history and said that he now has a profuse yellow discharge from his right ear and dizziness when he turns his head quickly, also that it was impossible for him to move the right side of his face or close the right eye. In other words, he had a complete facial paralysis.

Physical examination proved all organs normal.

Local examination showed a profuse discharge from the right ear from a cavity which seemed to involve the middle ear and mastoid. There was a complete facial paralysis on the right side.

Labyrinth examination showed no reaction to the caloric, or turning tests, and absolutely no hearing on that side. X-ray of the neck and lateral sides of the skull showed a large bullet, as will be seen in the accompanying X-ray photos.

A remarkable feature was that the right ear externally showed practically no signs of the bullet entering it. It appeared as if the marksman had used it for a bullseye and was 100 per cent perfect in his aim. A radical mastoid operation was advised, including exploration of the labyrinth. This was performed on May 23, 1927. The mastoid was found completely necrosed and only the distorted remnants of the semicircular canals could be outlined.

To our great surprise the bullet could not be seen nor felt. It has never been seen since, except on X-ray. The mastoid and labyrinth were completely curetted, a posterior flap was made, and the wound was packed. Treatment was continued until the wound was completely epidermitized about five weeks later.

Cerebellar Abscess, Sinus Thrombosis; Operation; Recovery. Dr. John McCoy.

Mrs. K., age 30 years, was seen in consultation with Dr. W. on Jan. 17, 1928, who gave me the following:

History: She complained of severe pain in the right ear and right side of her head about 10 days before. A myringotomy was performed by Dr. W. at that time. She reappeared several days later and said that, if anything, the pain in the head was much more severe and practically unendurable. The Doctor ordered X-ray pictures of the mastoids, which showed the left mastoid pneumatic and normal in appearance, the right mastoid sclerotic and cloudy.

She gave a history of an attack of acute ear trouble on the same side some two years before. The writer first saw this case in consultation on Jan. 17, 1928, after a simple mastoid had been performed by Dr. W. on Jan. 14, three days before. Dr. W. cleaned out a small sclerosed mastoid with granulations and pus in it. He found the sinus far forward. The sinus was unavoidably opened during this operation.

At the time the writer saw the patient three days after the operation the outstanding symptom was severe pain in the head, which was not relieved by the mastoid operation but which, if anything, was more severe. The temperature was 99°, the pulse was 84.

The labyrinth test with cold caloric showed that the labyrinth was active. Her mentality was heavy, which might be accounted for by the consistent doses of opiates which she was taking. Her powers of co-ordination, that is, finger to nose, and finger to finger, proved to be ataxic on the right side.

The cerebrospinal fluid was normal. Nystagmus was present and was peculiar in that in the course of a minute there would be perhaps three or four typical vestibular nystagmic movements of the eyes to the diseased side.

Eye Grounds: O. D. retinal veins slightly dilated, otherwise negative. O. S. fundus negative.

Diagnosis: It was felt that this patient had a deep epidural abscess in the region of the ductus endolymphaticus, causing vestibular irritation.

Operation: The mastoid wound was reopened by the writer, the dura exposed anterior to the sinus and well forward of the sinus. About one-half dram of epidural pus was found and removed. There was no apparent opening in the dura.

Subsequent History: The following day the patient appeared very bright and said that all headache had disappeared and took a normal interest in things about her. She did not require opiates. This continued for about three days, when she went into a state of coma. Examination of the cerebrospinal fluid showed that it was still normal.

Second Operation: On Jan. 22, the cerebellum was opened, not by the knife, but by pressure on a softened area with blunt anatomical forceps over the area where the epidural abscess had existed, and about 1 ounce of pus was removed from the cerebellum.

The temperature at this time was still 98° to 99°, the pulse 78 to 84. The blood picture was:

	R. B. C.	*Hgb.*	*W. B. C.*	*Poly.*	*Lymph.*
Jan. 17	3,240,000	62%	17,200	80%	20%
Jan. 27	3,040,000	60%	14,000	80%	18%
Feb. 9	3,640,000	75%	16,000	82%	18%

The cerebellar abscess was drained by guttapercha tissue and the patient's consciousness again returned to normal. Three days following this second operation the patient's temperature began and up-and-down course, from 99° to 106°, and while blood cultures proved negative, it was felt that she had a septic sinus thrombosis. Accordingly, a third operation was performed. The jugular vein was ligated and a series of about six blood transfusions were given her. The patient's temperature returned to normal and she progressed to an uninterrupted recovery.

The point the writer wishes to stress in this case is that we should always have in mind, when an ear begins to pain, is this a simple acute inflammation; or is it an acute exacerbation of a chronic condition?

DISCUSSION.

QUERY: Dr. McCoy spoke of curetting the labyrinth. I would like to know whether he curetted the labyrinth or did one of the standard labyrinthine operations. He spoke only of curetting the labyrinth.

DR. MARK J. GOTTLIEB: I would like to know whether the infection of the sinus was entirely due to the injury of the sinus during the operation or whether there might not have been some invasion of the sinus through the superior and inferior petrosal sinuses, because the cerebellar abscess and epidural collection of pus was found in this location. Direct infection of the lateral sinus by accidental tear during operation usually becomes manifest within 24 hours after such laceration and infection. The symptoms of lateral sinus thrombosis began in this case six days after the sinus had been punctured.

DR. MCCOY: There was no attempt made to do a typical labyrinth operation, for one could not see the outlines of anything resembling the labyrinth, except an occasional opening which might be the opening of the semicircular canal. The bullet had so shattered the labyrinth and caused necrosis of the bone that the only attempt was to get rid of all the necrosis and reach solid bone without attempting a labyrinth operation of any kind.

Answering Dr. Gottlieb, I feel that the infection of the bloodstream came from the original opening of the sinus. I think the clot remained quiescent for a long time and that it was during the two preceding operations the infection occurred. It finally lighted up about the tenth day following, producing a typical sinus thrombosis.

Pathology of the Ear in Meningitis (Illustrated by Lantern Slides). Dr. S. J. Crowe.

From the laboratory's collection of sections of the temporal bone from cases in which the principal cause of death was some form of acute meningitis, slides were selected illustrative of the variations in degree of involvement of several of the so-called "portals of entry". About 45 lantern slides of photomicrographs of parts of the internal and middle ears were projected and informally described.

One series showed material from persons with purulent meningitis without involvement of any modiolar structures, with perivascular inflammation only and with both perineural and perivascular reactions. Attention was called to the importance of the large perivascular spaces about the modiolar vessels. The variation from the normal, to complete necrosis of the soft tissues of the cochlear aqueduct, was shown in another series of slides. Infection of the endolymphatic duct and sac was observed only in material from persons with purulent labyrinthitis. Invasion from the middle ear through the membrane of the round window was illustrated by a series showing the normal, several degrees of inflammation and cases of complete perforation with labyrinthitis. Infection passing through the stapediovestibular articulation with and without necrotic changes in the ligaments was shown. The tissues of the fissura ante fenestram (Cozzolino's zone) contained numerous inflammatory cells along its

whole extent from the middle ear to the vestibule, on both sides, in an infant, age 6 months, indicating that this route may be not only a potential but an actual portal of entry for infections. Since the entire length of the fissure was included in each of several vertical sections, there could be no question of continuity.

DISCUSSION.

DR. WELLS P. EAGLETON: The New York Otologists are under a great obligation to Dr. Crowe for coming here, and I also wish personally to thank him.

Those of us who, in our younger days, acquired a little knowledge of the microscopic study of suppurative conditions of the labyrinth, know the great difficulties with which the subject is surrounded.

Within the last two years I undertook to compile all the cases of intracranial suppuration that had been followed by a complete microscopical examination in the laboratory under the direction of an expert researcher, and we found only 10 cases in the whole of the medical literature where patients have died as a result of suppuration, and then a complete microscopical examination had been made to follow the tract through which the infection had entered.

Turner and Reynolds have published six microscopic findings from the ear and nose. If my memory serves me rightly, four out of the six were from the nose; so you see how much this work has been needed, especially in this country.

It is a usual findng that meningitis — having originated from the ear and gone into the meninges—has gone in through a preformed way, and having reached the meninges has traveled through the blood vessels.

We know there are no lymph ducts in the lymph circulation, but we have learned clinically from postmortems of persons dieing of meningitis, the frequency with which there is associated a suppurative phlebitis which runs through the cortical veins. In Dr. Crowe's work we find confirmed in a general way that the method of infection, whether from the ear or otherwise, has been inwards from the blood vessels or lymphatics.

I cannot agree with him about the endolymph and the perilymph circulation. If you open a labyrinth when you get down toward the modiolus you get a seepage of fluid, and it seems to me that the endolymph and perilymph coming from the seepage itself is simply the normal way of filling these spaces.

Dr. Crowe did not speak of the importance of the arachnoid prolongations into the temporal bone. Last year in Paris I dissected eight cadavers that had been injected, and I was amazed to find that the arachnoid prolongations had not been accurately described, and all the descriptions are imperfect because the trabeculae had been torn. There are definite prolongations when the arachnoid goes from the brain into the bone. Every one of those is a potential point of infection.

Dr. Crowe has shown that the future of the surgical care of meningitis depends on our learning the pathology and histology of definite groups. I believe that when we can diagnose the exact site of entrance as we sometimes can now, say in the ductus lymphaticus cases, and recognize the internal prolongations that have been invaded in the particular cases, then this can be applied to the surgical treatment.

One of the greatest pleasure I have had in life was afforded me by a man I had never seen. He had done some very fine work on the vestibular canal and, to my amazement,—he was much younger than myself,—he said to me, "The work I have done came from the stimulation of the work you published in 1912."

Many things may happen to make Dr. Crowe realize how much the effort he has made in coming to New York and showing us the consummation of his effort has done to inform and inspire the otologists of America.

Again, I wish to thank Dr. Crowe for his extremely interesting presentation. He has left us "a light which doth not depart, and his look or a word he hath spoken wrought flames in another man's heart". You will have this to think of for some time to come, for such work as his is not likely soon to be supplanted.

DR. EDWARD B. DENCH: I do not think anyone can adequately discuss the magnificent paper which Dr. Crowe has just presented to us. It shows so much labor, so much thankless labor, for there is not very much glory in sitting down night after night in your laboratory preparing and examining microscopic slides and then, after a number of years of this sort of nerve-racking study, to come and talk these slides over for a period of a few minutes. The work that he has shown us tonight undoubtedly represents a number of years of painstaking investigation on his part.

Most of us have been almost entirely interested in the extension of inflammation from the middle ear into the labyrinth, and I am afraid that we are sometimes apt to forget that the inflammatory process may begin in the meninges, extend into the labyrinth, and thus involve the middle ear secondarily. While the extension in this direction is naturally very much less common than the other, the fact that it does occur should always be remembered. The slides showing this secondary inflammation of the middle ear certainly demonstrate the fact very clearly.

It is also interesting to note the great thickening which occurs about the oval window and about the round window in cases of both chronic and acute middle ear inflammation. I was also very much interested to learn of the occasional dehiscence which may occur over the promontory, in front of the oval and round windows. I had never heard of this dehiscence, although the speaker says that Seibeman refers to it as anatomical anomaly. Now that he mentions this, I believe I may have seen it in two cases of chronic middle ear suppuration in which a line cross the promontory in front of the oval and round windows. I had thought that this might be due to an injury, although there was no traumatic history in these cases. With so many possible avenues of invasion, and the frequency with which both acute and chronic suppurative otitis media occur, it seems rather remarkable that labyrinthine infection is not a more frequent complication of an inflammatory process within the middle ear. The sections show very clearly, however, that a barrier is thrown out between the middle ear and the labyrinth in all cases of acute and chronic middle ear suppuration. The enormous thickening of the mucous membrane in the middle ear, as shown by the sections, is the best demonstration of this barrier that I have ever seen. Those of us who have operated on many cases of acute and chronic middle ear suppuration are constantly seeing in the tympanomastoid cavity, instances where new bony tissue has been thrown out to protect the intracranial structures.

One very practical point which Dr. Crowe has demonstrated is the fact that if a labyrinth is invaded from without this does not necessaily mean that the entire labyrinth must be removed by surgical procedure, if operative interference becomes necessary to clear up the existing middle ear suppuration. It was beautifully demonstrated by the section which he presented how a fistula in one of the semicircular canals may completely wall itself off. Any interference with such a fistula would be to invite an acute suppuration of the labyrinth. It has always been my practice in this region to let sleeping dogs lie. A patient who has shown no labyrinthine symptoms, but whom we know is suffering from a dead labyrinth, may at the time of the operation present an eroded area in one of the semicircular canals. In the absence of labyrinthine symptoms, such an area should be simply walled off and no attempt should be made to explore the area with the probe. Nature in such a case has done her part in protecting the patient against infection through the cerebral spinal fluid. If we simply remove the primary area of infection from the middle ear by the radical operation and allow the eroded area in the semicircular canal to remain undisturbed. we have served the best interests of the patient. This is in direct opposition to the teachings of the German school of a few years ago, which emphasized the doctrine that in every case where we had a dead labyrinth, accompanied by labyrinthine fistula, the labyrinth should be removed.

If the condition is giving rise to symptoms, then remove the labyrinth at the time of the operation, if it is not and if compensation is taking place, let the labyrinth alone surgically, and allow the reparative process, which Nature has begun, to be undisturbed. I am sorry to say that I have seen serious infection result from too ardent su gical interference with the labyrinth in these cases.

It is most gratifying to have a paper of the kind to which we have just listened presented at this Academy meeting, which demonstrates how closely the microscopic changes which accur in these parts coincide with what some of us have learned through clinical experience. Many are rather loath to understand the great clinical value of a paper of this kind. Dr. Crowe possesses a great advantage, in that he is not only a trained clinician, but a trained pathologist as well. With him, clinical facts and micropathological findings go hand in hand.

We are greatly indebted to Dr. Crowe for the demonstration which he has given us. I doubt whether a more exhaustive demonstration of the points which he has cited has ever been made. He is to be congratulated on the magnificent work which he has done in this field and we should congratulate ourselves on the pleasure of having him with us.

DR. JOHN B. RAE: I wish only to express to Dr. Crowe my sense of the obligation due him not only from this Section but from American Otology in general. It promises well that we have men with enthusiasm enough to perform this tedious and exacting work. The microscopic sections were clear and illustrated each point made by the lecturer in demonstrating the pathways of infection.

DR. W. C. MCFARLAND: I can only express my personal appreciation of the demonstration.

DR. JOHN MCCOY: I also wish to thank Dr. Crowe for the very wonderful demonstration given. The practical point that occurred to me, aside from the very beautiful theory which he portrayed, is that the Doctor first showed us how the meningitis spread from the brain to the labyrinth and then from the middle ear and labyrinth to the brain. Of course these are all pathological specimens—all specimens from dead persons, and it has been my impression always that in those severe acute inflammations, such as follow scarlet fever and measles—infection from the middle ear—we had much better let the cases alone and not interfere surgically; yet the Doctor has shown us tonight where the inflammation has spread into the labyrinth, the meningitis has caused death. I wonder if in all these pathological examinations he had noted any reason to change his views in regard to that; whether it would be better to go in surgically and interfere, rather than to follow the rule. They tell us that 90 per cent of these cases spreading into the labyrinth, especially in childhood, get well without surgical interference.

DR. S. J. CROWE: I am not competent to answer Dr. Eagleton's question about the origin of endolymph and perilymph. There are, however, several anatomical and experimental facts that support the assumption that the endolymph, at least, is secreted by the cells of the Stria vascularis, flows through the cochlear duct—supplying nourishment and carrying away the waste products of the cells of the organ of Corti—then passes through the canalis reuniens and the saccule to the endolymphatic sac, where it is absorbed into the blood vessels.

The anatomical structures that support this assumption of the origin of the endolymph are:

1. The canalis reuniens is certainly open in the lower animals, and there is abundant histological evidence that it is open in man.

2. The organ of Corti has no known vascular or lymphatic supply—except the vas spirale under the basilar membrane.

3. The structure of the Stria vascularis and the large secretory cells described by Shambaugh furnish a physical apparatus that could supply the endolymph.

4. There is an abundant network of blood vessels[1] around part of the saccus and adjoining part of the ductus endolymphaticus that could absorb endolymph.

1. Guild, Stacy R.: Observations Upon the Structure and Normal Contents of the Ductus and Saccus Endolymphaticus in the Guinea Pig. *The Amer. Jour. Anat.*, Vol. 39, 1-56, 1927.

These anatomical suppositions are borne out by the experimental work of Guild[2], who introduced a solution of potassium ferrocyanid and ironammonium citrate into the cochlear duct of living guinea pigs. The animals were killed after intervals of from 45 minutes to 44 hours. In microscopical sections the Prussian blue granules, which are precipitated by the addition of acid to the fixing fluid, were found in the ductus and saccus endolympmphaticus; these granules were not merely in the lumen but had apparently been excreted and were found in the walls and perivascular spaces.

These experiments indicate that in the guinea pig, at least, there is a normal flow of endolymph from the cochlear duct to the saccus endolymphaticus. The cochlear aqueduct provides a direct communication between the perilympahtic spaces in the cochlea and the subarachnoidal space.

In answer to the question about acute infections: I think one of the fundamental facts of special, as well as general surgery, is that surgical therapy should be postponed as long as possible in the presence of an acute infection. I perhaps did not make it clear that the slides I showed tonight were nearly all from cases that had a primary meningitis with secondary involvement of the inner ear. In only two or three cases did the history and clinical course suggest a primary middle ear infection as the source of the meningitis.

2. Guild, Stacy R.: The Circulation of the Endolymph. *The Amer. Jour. Anat.*, Vol. 39, 57-81, 1927.

BOOK REVIEW.

Diseases of the Throat, Nose and Ear. By Dan McKenzie, M.D., F.R.C.S.; President, 1926-27 Section of Otology, Royal Society of Medicine; Surgeon, Central London Throat and Ear Hospital; Oto-Laryngologist to the French Hospital, London, etc. Second Edition, two volumes. St. Louis: C. V. Mosby Company, 1928. Price $17.00.

We agree with the first sentence by the author in the preface to the first edition of his book wherein he states: "This book has been written from the practical point of view and for that reason many interesting scientific problems and issues have necessarily been omitted. On the other hand, considerable attention has been devoted to operative surgery, but here, also, limitation of space has rendered it impossible to describe the many modifications and variations of the operation."

In the second edition, just issued, the same plan has evidently been adopted and the practical consideration, operative technique and essential treatment dominate the two volumes which the additional data during the past eight years have made necessary.

It is an excellent working manual of applied Oto-Laryngology eliminating unnecessary verbiage, incorporating the operative experiences of the author, adding in brief text and choice illustrations, selective and rare cases of colleagues and presenting the entire subject-matter in a clear and concise form.

The outstanding features of this two-volume textbook are the comprehensive character of the subject-matter as presented throughout the range of practical Oto-Laryngology and the unusually fine illustrations in black-and-white and in colors of the selective subject-matter which the author puts forth. These features, together with good judgment, long experience and simple, graceful style of expression, constitute the strength and value of this treatise.

M. A. G.

PUGET SOUND ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY.

Regular Meeting, Sept. 18, 1928.

The regular meeting of the Puget Sound Academy of Ophthalmology and Oto-Laryngology was held Tuesday, Sept. 18, 1928, at the Medical and Dental building, Seattle. In the absence of Dr. E. C. Wheeler, the President of the Society, Dr. W. O. Bell, the First Vice-President, presided.

The resignation of Dr. E. C. Wheeler as President of the Society was read, and accepted. A committee composed of Drs. Washburn, Clark and Waltz was appointed to write Dr. Wheeler a letter expressing appreciation of the work done in the Society and wishes for a speedy recovery from the ill health which necessitates the resignation.

The coming Pacific Coast meeting was discussed by several members and, on motion of Dr. Dowling, the Secretary was asked to get in touch with the officers of the Coast Society and see if it would not be possible to have the date of the Coast meeting set before or after the Denver Lectures so that the members may attend both without so much traveling and loss of time.

Operative Cure of Chronic Suppurative Maxillary Sinusitis. Dr. J. H. Harter.

Dr. Harter gave a resume on his paper on Operative Cure of Chronic Suppurative Maxillary Sinusitis. The rhinologist should impress the general physician with the frequency of chronic suppurative maxillary sinusitis. These infections should be sought for as are now infections of teeth and tonsils.

In diagnosis within the specialist's office, every routine case should be transilluminated. If this or intranasal examination is at all suggestive of sinus infection, a series of X-ray pictures should be taken. Any suspicious antrum should be irrigated. X-ray plates taken after lipiodol irrigations, also viewing the interior of the cavity with an antroscope, is being practiced by some men, but has not as yet come into general use.

If diagnosis of chronic disease of the antrum is made and if the case does not respond to simple treatment, I favor the Denker operation, local anesthesia, for the following reasons:

1. The patient can be assured that unless complications arise, he can leave the hospital in four days, return to work in 10 days, and that after healing, the cavity will be no more subject to infection than is the lining membrane of the nose.
2. At any time following operation, a straight applicator or cannula can be inserted into the cavity and irrigation will demonstrate to the patient that no infection is present.
3. Hemorrhage and shock are minimized.
4. The postoperative period is shortened and painful after-treatments are avoided.

DISCUSSION.

DR. J. S. DAVIS: We practically always do an internasal operation as we find very few cases that require anything more radical. We do not irrigate after the operation as we feel that irrigation tends to prolong the convalescence. Quite often, after removing the tip of the inferior turbinate, if bleeding is very troublesome, we use a silver nitrate caustic applicator on the bleeding margin and get a drier field.

We recently had a case of atrophic rhinitis resembling ozena, in that there was odor, crusts and pus. This patient had had a double antri operation several years ago, and both antri were open. About three months ago, she had a goiter operation and after one week her nose cleared up and we have been unable to demonstrate any crusts or pus in her nose since that time. This has brought up the question in our minds as to the relation of thyroid to ozena.

DR. CLARK: I feel that Dr. Perry not being present, I must take his part, as the intranasal maxillary operation is also my operation of choice. In my hands, this operation, as far as I know, has given 100 per cent results in a good series of cases. As to it being called the major, and the Denker the minor operation, I have surely not found it so, as the average time loss in my cases has averaged two days, whether it be a uni or bilateral case.

The after-treatment is very little and if one is kind, he does not leave a mutilated nose. Give one man a hammer and nails and boards, and he produces a fine piece of furniture; another man just wastes boards and nails. And so it is with the various operations, and why one prefers an operation that he does well and successfully to a perhaps more classical one that he just doesn't do so well. This subject could be discussed profitably all night, and we wouldn't know when to quit.

Relief of Traumatic Facial Paralysis by Operation. Dr. J. T. Dowling.

Patient age 28 years, male, single, sailor. Fell in hold of ship, Feb. 15, 1928. Rendered unconscious for three days. Treated for fractured skull in Latouche, Alaska. Complete right side facial paralysis since date of accident. Discharged from Latouche Hospital, March 4, 1928, for transfer to Seattle. Admitted to Virginia Mason Hospital, March 10, 1928. Unable to demonstrate fractured skull with X-ray. Other laboratory findings negative. Physical findings from our standpoint were: Complete right-sided facial paralysis. Patient unable to close right eye or draw his mouth to the right, and his nose was markedly drawn towards the left side. In looking into the right external auditory canal we could not see the entire tympanic membrane, as our view was obstructed by what we thought was a spicule of bone in the region of the facial canal.

The patient was treated by us and with ultraviolet ray, with no apparent improvement of the facial paralysis. The patient finally consented to have a mastoid operation.

On May 22, 1928, a radical mastoid operation was done. At this time we were able to demonstrate a fracture through the mastoid bone and our diagnosis was confirmed regarding the displacement of the facial canal forward into the external auditory canal. The spicule of bone was removed and the nerve exposed and all pressure released. The nerve was badly traumatized and hemorrhagic, but, in our opinion, not severed by the fracture.

After completing the operation, we handled the case as an ordinary radical mastoid. Patient discharged from the hospital on June 13, 1928, after an uneventful recovery.

Since his discharge from the hospital, we have been observing the patient several times a week, and having him treated with ultraviolet ray and massage. Much to the delight of all parties concerned, we are now seeing a gradual recovery from mouth upwards, the nose now being almost straight.

Conclusion: In my opinion, this case is one of facial paralysis due solely to pressure from a fracture through the mastoid bone.

Suture of Tenon's Capsule in Enucleation. Dr. J. T. Dowling.

Patient age 45 years, laborer. Aug. 3, 1928, struck in left eye with a large hook. Upper left lid lacerated. Eyeball lacerated through limbus, with loss of vitreous. X-ray, left eye, for foreign body negative. Wassermann negative. Urine negative. Admitted to hospital, Aug. 3, 1928. Treated with antiseptics daily. Enucleation done Aug. 16. Closure. Tenon's capsule closed with catgut. Conjunctiva clased with same. Discharged from hospital, Aug. 27. Treated daily in office. Artificial eye inserted, Sept. 10. Results satisfactory.

Dr. A. E. Hillis, *Secy.*

IN MEMORIAM.

DR. GREENFIELD SLUDER.

Dr. Greenfield Sluder, born in St. Louis in 1865, died October 9, 1928, at Barnes Hospital after a protracted illness, which began in Boston more than two years ago and which culminated in femoral thrombosis, amputation of the right leg, and pneumonia.

Dr. Sluder was a product of St. Louis, educated at the Manual Training School there and graduated from St. Louis Medical College in 1888. He served four years as interne and senior resident at the St. Louis City Hospital and practiced general medicine for six years. This was followed by post-graduate training in Rhinology and Laryngology abroad. He returned to St. Louis well equipped to enter his specialty of Rhino-Laryngology and

almost from this first period was active in the teaching faculty of the Laryngological Department of Washington University School of Medicine. He was promoted to several positions in this department until in 1922 he was appointed Clinical Professor of Oto-Laryngology.

The outstanding characteristic of this man was his talent and ambition for scientific investigation and research and in the successful development of this talent he probably has had no superior among our colleagues in American Laryngology. His was the mind of the carefully trained and painstaking student absorbed in the study of unsolved problems and anatomical details and to these factors more than any other in his splendid career must be attributed his especial accomplishments. His original technique in the enucleation of the faucial tonsil, contribution to the paranasal sinuses, studies of the spheno-palatine ganglion, the physics and treatment of vacuum headache,—all may be summarized as the output of his fine investigative mind, his knowledge of physics and mechanics and his altruisms as a pure scientist.

He was an indefatigable worker in Laryngology and his contributions to this specialty are among the best and enduring legacies to American Laryngology. The three outstanding monographs which he published are: "Concerning Some Headaches and Eye Disorders of Nasal Origin"; "Tonsillectomy by Means of the Mandible and a Guillotine"; "Nasal Neurology, Headaches and Eye Disorders".

In 1926, Dr. Sluder was honored with the Presidency of the American Laryngological Association but the unfortunate occurrence of his illness about this time made it a regrettable incident that he was unable to preside at the Annual Meeting of the Fellows on this occasion.

In the death of Dr. Sluder, American Laryngology has lost one of its ablest and scientifically useful members. The quality and substance of his investigations and conclusions will remain an integral part of our literature and practice for an enduring period.

To his family we extend our sincere sympathy. M. A. G.

HOLGER MYGIND.

Holger Mygind died on October 1, 1928, in the Kommune Hospital in Copenhagen in his 73rd year. He died in the department that he himself organized and to which he was so attached. Mygind had suffered many infectious diseases in the past few years, but with his unswerving energy he forced himself to continue the pace of his younger days and only the closest observer could notice that his health was broken. Only by special efforts was he enabled to serve as vice-president of the First Inter-National Oto-Laryngological Congress and at the banquet he astounded his hearers with the energy of his speech.

In Mygind, Otology has lost one of its foremost pioneers and the Danish Otologists a very good friend. A strong, energetic and upright personality has passed away and has left a gap which will not be easily filled.

Mygind graduated in Copenhagen in 1880 and served in the Garnison and Kommune Hospitals. In 1884, he became an assistant to Wilhelm Meyer, and in 1898 he was appointed chief of the Ear, Nose and Throat Clinic of the Kommune Hospital. He reorganized the Clinic and in 1923 was succeeded by his son, Sydney Mygind.

Mygind earned an enviable reputation not only as a doctor and scientist but also as a teacher.

One of his earliest and best works was the investigation of congenital deafness, on which he wrote a monumental monograph. He was a co-worker of Finsen in the investigations on light-therapy and wrote numerous articles on the treatment of lupus of the upper respiratory tract. He was also one of the first advocates of early operation in cases of otitic meningitis and presented a paper on the subject to the British Medical Association in 1922.

Mygind took an interest in speech defects and it was due to his efforts that in 1898 the state organized an institute to deal with these cases. It was due also in no small measure to Mygind that in 1911 the first Northland Oto-Laryngological Congress was held. He was president of the Danish Oto-Laryngological Society in 1901-1903 and 1916-1918.

When he retired from the Kommune Hospital in 1923 he devoted more time to his hobby, archeology, and spent all his vacations in his beloved Pompeii. He delivered many lectures on this subject.

To his co-workers and assistants, Mygind was always a good and true friend, and was always gracious in giving advice and help.

N. Rh. Blegvad.